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The Journal of Periodontology
is dedicated to
Dr. Gillette Hayden
and
Dr. Grace Rogers Spalding
for their pioneer efforts and
selfless devotion in behalf
of periodontia and the
American Academy
of Periodontology

On February 10, 1951, the Council of the American Academy of Periodontology unanimously voted to recognize the value of the services of Dr. Grace Rogers Spalding by dedicating the Journal of Periodontology to her. In the future, therefore, this dedication page will carry her name as well as Dr. Gillette Hayden.

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The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as papers submitted directly to the Editor.

Thirty-fifth Annual Meeting

THE AMERICAN ACADEMY OF PERIODONTOLOGY

October 11, 12, and 13, 1951

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Olin Kirkland, D.D.S.

Montgomery, Alabama

The honorary degree of Doctor of Science was awarded to Dr. Olin Kirkland of Montgomery, Alabama, by the University of Alabama on February 3, 1951. In conferring this degree, Dr. John M. Gallagher, President of the University of Alabama said:

"Olin Kirkland, native and citizen of Montgomery, you have rendered dental service to your fellow citizens for almost half a century. A pioneer in the development of the dental specialty of periodontology, you have come to be recognized as one of its eminent authorities. From your vast clinical experiences you have devised many original dental surgical procedures. These you have unselfishly passed on to your fellow practitioners, and to students by your many articles in dental journals and textbooks, and by your lectures and teaching. Recognizing the importance of dental education you have served for many years as a member of the Advisory Board of your Alma Mater, the Emory University School of Dentistry, and have championed the establishment of a dental school as an integral part of the University of Alabama. By these accomplishments you have brought honor to your native State and have contributed significantly to the progress of your profession and to the relief of mankind."

THE JOURNAL OF PERIODONTOLOGY

APRIL, NINETEEN HUNDRED FIFTY-ONE

Experimental Periodontal Reattachment in Rhesus Monkeys*

By SIGURD RAMFJORD, L.D.S., M.S., PH.D. *Ann Arbor, Michigan*

REATTACHMENT, in periodontal terminology, means a unification or an attachment between the tooth and the adjacent soft tissue wall of the periodontal pocket. It has, for years, been one of the most controversial and extensively debated subjects in the periodontal literature.[8, 10, 26, 31, 32] An attachment of the surrounding soft tissues to the tooth, following treatment of the periodontal pocket, has clinically been obtained by a number of outstanding periodontists and teachers of periodontia.[2, 4, 5, 6, 7, 13, 14, 15, 18, 19, 25, 29, 35, 36, 37, 40] The convincing histological proof, however, has been missing for the obvious reason that the fortunate patients on whose teeth the healing has been obtained did not want to sacrifice their teeth for microscopic examination.

Two experimental attempts on reattachment in rhesus monkeys have been reported. One was reported by Stone[38] who surgically created periodontal pockets and left them alone. He obtained some healing in the bottom part of the pockets. Similar experiments with surgically made pockets and some ensuing healing have been reported both in other animals and in man.[3, 6, 12, 27, 34]

Fish[16] reported another experiment with surgically made pockets in rhesus monkeys. He obtained only some degree of healing, or what he called reattachment, in cases where the gum margins were healthy, but he did not obtain any reattachment where a gingivitis existed. When the gingiva was inflamed, the epithelium simply grew down to the point where the periodontal fibers were still normally attached to the uninjured cementum.

OWN INVESTIGATION

The first purpose of this experimental work was to investigate the possibility of reattachment in inflamed epithelialized periodontal pockets in rhesus monkeys by using a therapy which has proved to give clinical reattachment in man.[2, 14, 15] The second and most important object of the investigation was to study the nature of the reattachment if any was achieved.

The experiments were performed on four healthy rhesus monkeys from four to six years old. A gingivitis, ranging in degree from mild to severe, was present around the teeth that were included in the experiment, but no periodontal pockets were present. Kodachrome pictures were taken (Fig. 1). The distances

*From the Dental School, University of Michigan, Department of Oral Pathology and Periodontia.

*This is a portion of the material from a thesis submitted in partial fulfillment of the requirements for a Ph.D. degree.

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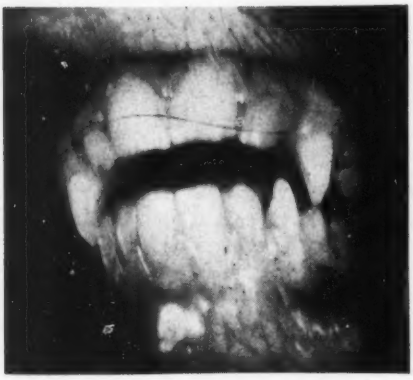

Fig. 1. Monkey #1868 before the experiment.

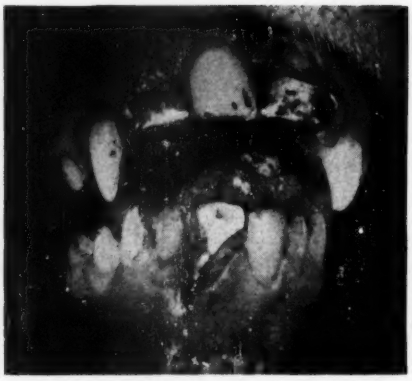

Fig. 2. Monkey #1868, the copper bands in place.

from the incisal corners of the incisor teeth to the bottom of the interproximal gingival crevices were measured with a thin silver point of the type which is used for root canal fillings. These teeth had sharp, well-defined mesial and distal incisal corners, to provide definitely fixed points on the teeth from which the height of the interproximal epithelial attachment and later the decrease in depth of the pockets could be measured. In many previous publications on reattachment the depth of the pockets has been measured from the free gingival margin which is continually changing in height during the treatment. This has resulted in a false interpretation of experimental data. When mesial or distal measurements are given in this paper without further designation, they always refer to the distances from the mesial or distal incisal corners to the bottom of the interproximal crevices. The monkeys were anesthetized by nembutal (pentobarbital sodium), and deep interproximal pockets extending below the alveolar crest margin were created. The periodontal membrane is rather thin in monkeys, so some alveolar bone had to be removed in order to obtain pockets of sufficient width and depth, and in most cases some tooth substance also was removed at the bottom of the created pocket for futher reference. Copper bands were adapted to the experimental teeth and extended down to the bottom of the surgically created pockets (Fig. 2). A similar method has been described by Swenson.[39] He adapted and polished the bands as close to the roots as possible to minimize the irritation, but in this experiment a severe inflammation was desired, so the bands were cemented on the teeth without being closely adapted. Occlusal interference was avoided, but nothing was done to eliminate the interproximal trauma which occurred from forcing the band through the tight interproximal contacts. The interproximal measurements of the bands were recorded. In a few cases the bands did not extend quite to the bottom of the surgically created pockets, as may be seen in Table I. Swenson, in his experiment on dogs, removed the copper bands after fifteen days. Tissue sections at that time showed inflammation and downgrowth of epithelium on the gingival wall to the bottom of the pocket. The dogs were sacrificed 150 days after the bands were removed, and histologic sections showed a pocket very similar to the pathologic periodontal pocket occurring in man. He examined one hundred pockets of this type and stated that no reattachment occurred after the bands were removed. According to his findings, this type of copper band, cemented on the teeth for fifteen days in dogs with a healthy gingiva, invariably created permanent periodontal pockets. Fish[16] observed in monkeys that if a gingivitis was present before the surgical

Fig. 3. Monkey #1868. At the time of death.

pocket was made, the inflammation from the gingivitis alone would prevent any reattachment.

A severe acute gingival inflammation developed shortly after the bands were cemented, but the appearance gradually changed to resemble very closely the clinical picture of a periodontitis caused by a gold crown with overhanging margins. It was decided to leave the bands on the teeth for as long as twenty-six to thirty-one days in order to assure time enough for the epithelium to grow down the gingival wall to the bottom of the pocket, and for the inflammation to assume more of the chronic character seen in periodontitis. The irritation from the bands and the destruction of transeptal fibers caused pockets to develop on the adjacent teeth, so in the cases in which only one interproximal pocket was made on a tooth, a pocket developed gradually, both on the adjacent tooth and on the opposite interproximal side on the same tooth. These pockets were not surgically created, but pockets developed as a sequence of inflammation. This proves what has been assumed from clinical evidence that irritation and inflammation may lead to pocket formation. Five of the deepest pockets of this type are included in the report. Kodachrome pictures were taken when the bands were removed, and the distances from the incisal corners to the bottom of the pockets were recorded. The teeth were scaled lightly and the pockets were washed with a mild iodine lotion. This treatment was repeated after two and after seven days. Ten days after the bands were removed the inflammation had subsided somewhat, but with the poor oral hygiene and the accumulation of food debris and materia alba on the teeth and in the pockets, an active inflammation with some accumulation of pus persisted. No changes in the depth of the pockets could be observed. The roots were thoroughly scaled and filed as smooth as possible. Thereafter, the pockets, both artificially made pockets and pockets gradually developed from local inflammation, were packed with cotton strings that

Fig. 4. Monkey #1899, tooth #8 distal. Clinically 3 mm. decrease in pocket depth following treatment. The white line indicates the depth of the experimental pocket and the previous position of the copper band.

TABLE I.

Clinical Findings in Reattachment Experiment

Monkey No.	*Teeth No.*	*Measurements (mm.)*					*Healing before removal of bands (mm.)*	*Reattachment following treatment (mm.)*	*Bands Present (Days)*	*From removal of bands to death (Days)*
		Before Operation	*After Operation*	*Bands*	*Bands Removed*	*At Death*				
1887	8 mes.	6.0	10.0	9.0	8.5	7.5	1.5	1.0	26	114
1887	8 dist.	5.5	9.0	8.0	8.0	5.5	1.0	2.5	26	114
1887	24 mes.	5.5	10.0	10.0	10.0	7.5	0.0	2.5	26	114
1887	24 dist.	6.0	10.0	9.0	9.0	7.0	1.0	2.0	26	114
1868	8 mes.	5.0	10.0	9.0	9.5	7.5	0.5	2.0	28	114
1868	8 dist.	4.5	Not oper.	.....	8.0	4.5	0.0	3.5	28	114
1868	10 mes.	4.0	8.0	8.0	7.5	4.5	0.5	3.0	28	114
1868	9 dist.	5.5	Not oper.	.....	8.5	6.5	0.0	2.0	28	114
1868	24 dist.	3.0	10.0	10.0	Band lost	5.0	2.0	3.0	5	137
1899	8 dist.	5.0	11.0	11.0	11.0	8.0	0.0	3.0	31	117
1899	10 dist.	3.0	10.0	10.0	10.0	8.5	0.0	1.5	31	117
1899	10 mes.	5.0	Not oper.	.....	7.0	5.0	0.0	2.0	31	117
1899	24 mes.	6.0	11.0	11.0	11.0	9.0	0.0	2.0	31	117
T.28	8 dist.	4.0	10.0	10.0	10.0	10.0	0.0	0.0	26	90
T.28	7 mes.	3.0	Not oper.	.....	6.0	4.5	0.0	1.5	26	90
T.28	10 mes.	3.5	9.5	9.5	9.0	6.0	0.5	3.0	26	90
T.28	24 mes.	4.0	10.0	10.0	10.0	8.0	0.0	2.0	26	90
T.28	25 mes.	4.0	Not oper.	.....	8.0	4.0	0.0	4.0	26	90
T.28	23 dist.	4.0	8.0	No band	.....	4.0	4.0 (Healing)	.....	.....	.....
T.28	13 dist.	2.0	7.5	No band	.....	2.0	5.5 (Healing)	.....	.....	.....

had been saturated in a mixture of 75% phenol and 25% camphor. The packs were removed after five minutes and the pockets flushed with warm water and later dried with cotton rolls and hot air. An attempt was made to scrape away the cauterized epithelial lining and to get blood to fill the pocket. The area was kept dry for about ten minutes to allow the blood to clot within the pocket before it was exposed to the saliva. The teeth were later scaled and polished twice a week until the monkeys were sacrificed. The phenol-camphor pack treatment was repeated one month later. The first scalings after this type of treatment have to be performed with great care because the developing attachment is easily destroyed at that early stage. Materia alba accumulates very fast on the teeth of the monkeys and the clinical results were not as good as the results obtained in cooperative patients. A mild gingivitis was still present in all cases at the time of death. The monkeys were sacrificed 90 to 117 days after the bands were removed. Kodachrome pictures (Fig. 3) and dental x-rays were taken, clinical findings and measurements recorded, models made, and the jaws immediately fixed in Bouin's solution.

CLINICAL FINDINGS

The clinical findings are recorded in Table I. In the few instances where the bands did not extend quite down to the bottom of the surgical pockets, a healing occurred before the bands were removed, reattaching the soft tissues up to, or almost up to the edge of the band. In a few cases the measurements at the time of removal were found to be 0.5 mm. shorter than the bands, which can be explained by the bands being situated at some small distance from the root, thus allowing a limited healing to take place in the space between the edge of the band and the root. One should also allow for a possible error of 0.5 mm. in the clinical measurements of this type. The measurements were always taken along the root surface and could, therefore, in these cases be slightly shorter than the bands. The photomicrographs shown in this article are all from cases in which no healing had occurred before the periodontal treatment with removal of the epithelial lining had been performed. The clinical reattachment is equivalent to the difference in measurements from when the bands were removed to when the monkeys were sacrificed. Reattachment measuring from one to four mm. was obtained in all but one of the eighteen pockets which were treated.

In a few additional instances pockets were created surgically and left alone. Two such cases are included in Table I. They showed complete healing which is in accordance with previously mentioned reports.

MICROSCOPIC FINDINGS AND INTERPRETATIONS

In most cases it was possible from healed surface injuries on the root to identify microscopically the original depth of the surgically created pockets (Fig. 4). Fragments of cementum and dentin were incorporated between the periodontal fibers, and new cementum appeared to be deposited on these displaced parts of the root (Fig. 5b). The zone of reattachment, from the bottom of the created pocket to the bottom of the gingival sulcus, showed both attachment of connective tissue fibers associated with formation of new cementum and epithelial attachment on old cementum or dentin surfaces[1, 11] (Fig. 6a). Evidence of resorption and subsequent healing was often observed on the previously denuded root surfaces (Figs. 6b and 6c). Alternating zones of connective tissue and epithelial attachment were observed in some cases (Figs. 7a and 7b). From correlating clinical and histological data it was evident that both connective tissue and epithelial reattachment had occurred on root surfaces which had been, first, covered by bands and later exposed in chronically inflamed periodontal pockets (Fig. 5a). New bone formation was often noted on the alveolar crest (See Figs. 5a and 6a). The new connective tissue attachment to the root was always associated with

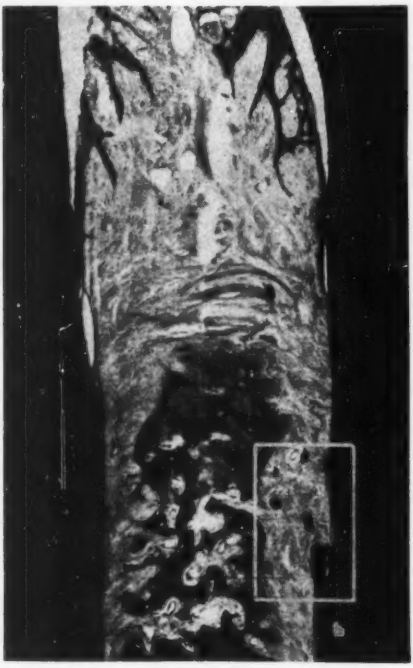

Fig. 5a.

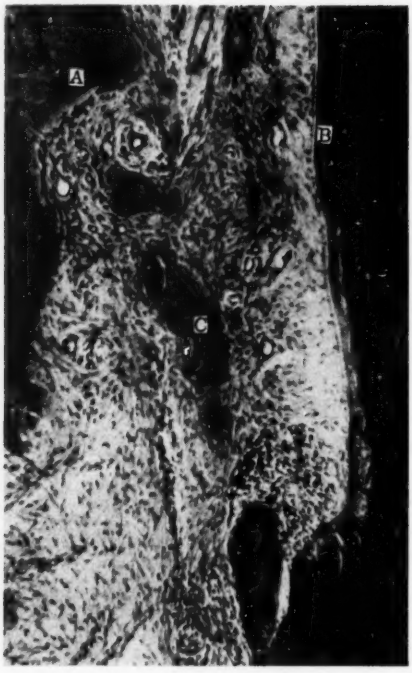

Fig. 5b.

Figs. 5a and 5b are from Monkey #T28, tooth #10 mesial. Clinical decrease in the depth of the pocket 3.5 mm. during treatment. Deposition of bone on the alveolar lamina (A). Cementoblastic activity on the root (B) and on the surface of dislodged fragments of the tooth (C).

cementoblastic activity (Figs. 8a and 8b), and in some places a rather thick layer of new formed cementum was observed.

The new epithelial attachment resembled the normal epithelial attachment, the nature of which is not known in detail.[9,30] The epithelial reattachment often consisted of only a few layers of flat almost spindle-shaped cells (Fig. 9), in other places the epithelium was growing out in deep pegs. Numerous nests and strands of epithelium were seen in the area of healing, extending deep in the connective tissue. They were possibly remains of epithelial pegs from the time that the epithelial covering of the pocket was cauterized and scraped away (See Fig. 7b). A zone of concentrated accumulation of inflammatory cells, mainly plasma cells, but also some lymphocytes and mononuclear histiocytes, were often seen adjacent to the epithelial reattachment (Fig. 10). Infiltrations of inflammatory cells were occasionally observed between the fiber bundles of the reattached connective tissue, extending down to the bottom of the previous pocket (Fig. 5b). A mild gingivitis was present in all of the cases that had been treated.

No reattachment occurred from the time the bands were removed until the epithelial lining of the pocket was cauterized and scraped away. In the one case that didn't show any diminishing of pocket depth during the period of treatment (Fig. 11), a very marked inflammation was persisting to the time of death of the animal. The pocket was narrow and the treatment had obviously not extended to the bottom of the pocket. The degree of organization of the blood clot in the pocket was probably

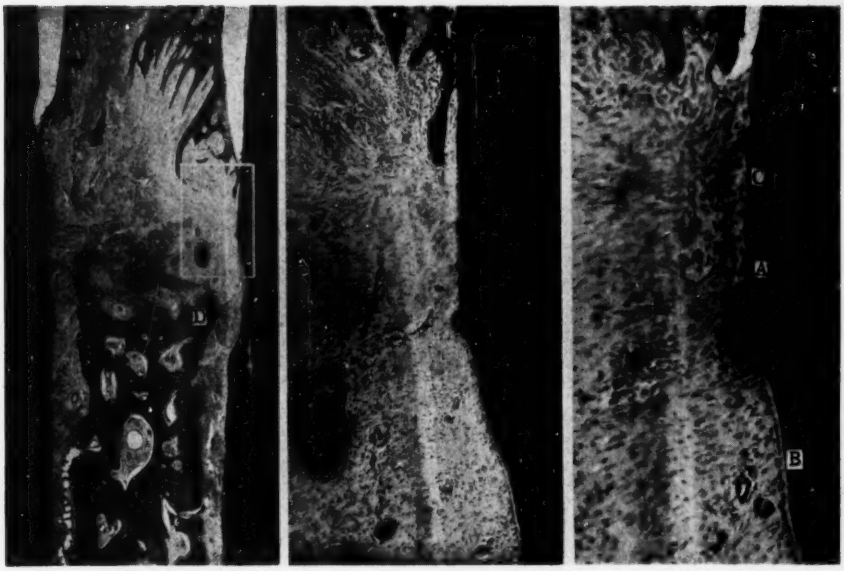

Fig. 6a. Fig. 6b. (x105) Fig. 6c. (x320)

Figs. 6a, 6b, and 6c are from monkey #1899, tooth #24 mesial. Clinically the depth of the pocket decreased 2 mm. during treatment. Resorption and repair of old cementum (A). New formation of cementum both on the previously denuded dentin (B) and on the surface of the old cementum (C). New bone formation reestablishing the normal width of the periodontal membrane (D).

dependent upon the amount of irritation and inflammation which was residual following phenol-camphor treatment. The cauterization and the removal of the epithelial surface will produce an inflammatory reaction, and the organizing blood clot will be effected by local irritation, but it was believed that the state of the underlying tissue before the attempt on reattachment was the most important factor, this also conforms with the repeated statement in the literature that reattachment should only be attempted when the local inflammation has subsided. Epithelium from the free gingival margin starts to cover the organizing blood clot, and when the proliferating basal layer of the epithelium reaches the position in which the granulation tissue contacts the tooth, it grows down along the root surface, attaching itself to the tooth until its downgrowth is stopped by connective tissue fibers attached to the root surface by new formed cementum. This process is similar to the downgrowth of the epithelial attachment which occurs both during the physiological eruption and pathological pocket formation. It is no reattachment of old epithelium, but new growing epithelium in which tonofibrils utilize the microscopic roughness on the tooth surface for attachment.

It is frequently stated in the periodontal literature that the position of the epithelial attachment is determined by the presence of periodontal fibers, and that following a destruction of fibers the epithelial attachment will grow down until it reaches attached fibers.[17, 20, 21, 23, 24, 33] It appeared as if the reattachment of the epithelium followed the same pattern as the normal epithelial attachment. A fibrous organization of the granulation tissue in the area of repair and cementoblastic activity appeared to be dependent upon the severity of

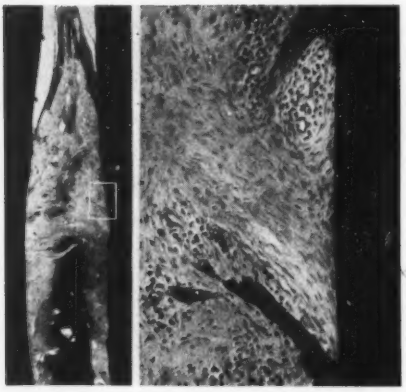

Fig. 7a. Fig. 7b. (x320)

Figs. 7a and 7b are from monkey #1887, tooth #24 distal. Alternating zones of connective tissue and epithelial reattachment. Inflammatory infiltrations, chiefly plasma cells.

the inflammation present. If the granulation tissue that replaced the blood clot was pyogenic in type the environment was not conducive to cementoblastic activity nor to production of collagenous fibers,[22, 28] and there was nothing to prevent the epithelial attachment from growing down along the root surface until it reached the residual periodontal fibers at the bottom of the previously produced pocket. The practical application of the reported findings should be that subgingival curettage attempting reattachment of connective tissue type is only indicated where the periodontal tissues can be brought to a good state of health during the hygienic phase of the periodontal treatment.

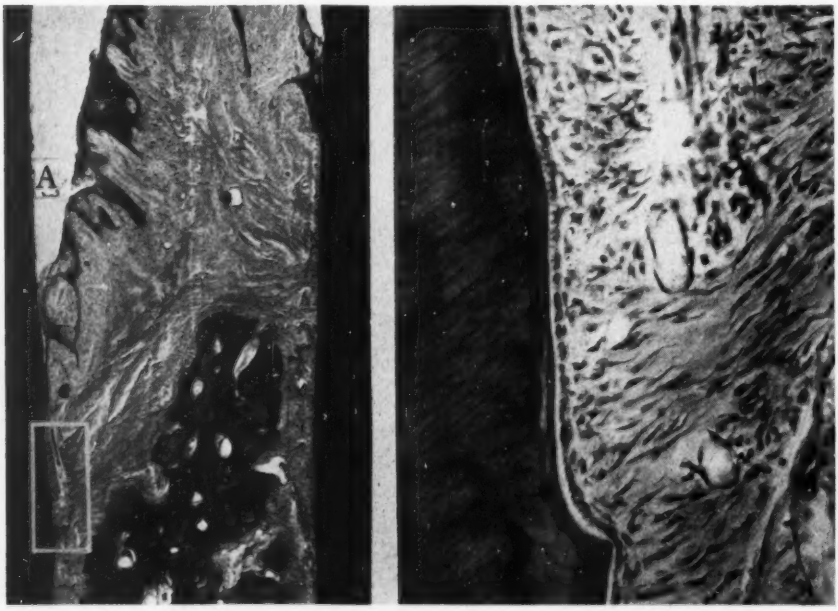

Fig. 8a. Fig. 8b. (x280)

Figs. 8a and 8b are from Monkey #T28, tooth #24 mesial. Clinically 2 mm. reattachment during treatment. Residual pocket (A). About one-half of the zone of reattachment is connective tissue with newly formed cementum (8b). No evidence of inflammation in the area of connective tissue reattachment.

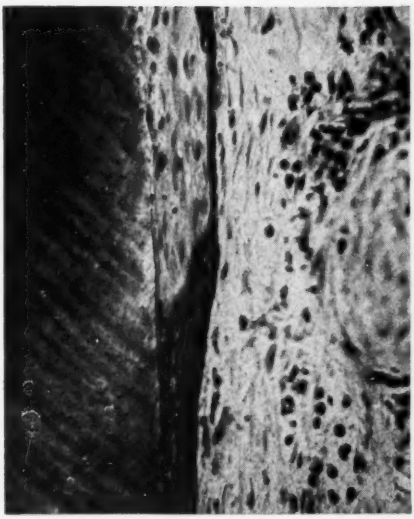

Fig. 9 (x320). Monkey #1899. Tooth #10 distal. Epithelial reattachment, spindle-shaped squamous epithelial cells on previously denuded dentin surface.

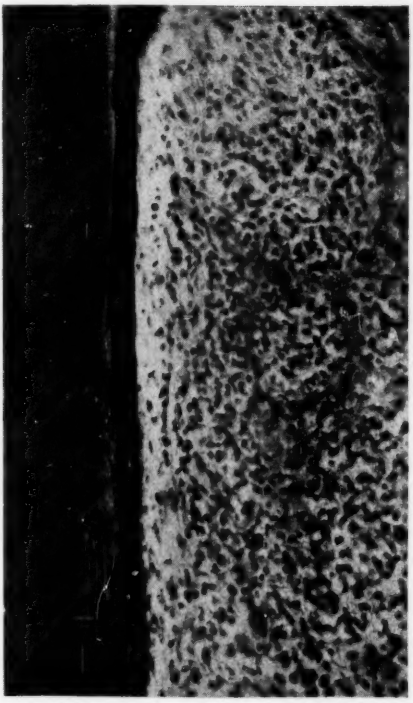

Fig. 10 (x320). High magnification of Fig. 4. Epithelial reattachment in the presence of severe inflammation.

CONCLUSION

1. Both connective tissue and epithelial reattachment was observed on root surfaces that had been exposed in an inflamed periodontal pocket.

2. Removal of the epithelial lining of the periodontal pocket was necessary to obtain reattachment.

3. The degree of inflammation in the area of healing appeared to determine whether the reattachment would be epithelial or connective tissue in type. The least degree of inflammation was associated with connective tissue reattachment.

4. In one case of severe gingival inflammation no reattachment was obtained.

The author reports he is indebted to the Department of Epidemiology, School of Public Health, University of Michigan, for providing the facilities for this work, and also to Miss Edna K. Mallory, School of Dentistry, for the preparation of histological sections, and to Miss Winnifred Arnold, School of Dentistry, for photographic services.

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Fig. 11. Monkey #T28, Tooth #8 distal. Clinically no reduction in depth of the pocket. Intraalveolar pocket extending slightly below the level of surgical detachment (A).

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Significant Considerations in the Prognosis of Periodontal Disease

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ONE of the basic requirements in clinical periodontology is the ability to predict with some accuracy just what will be the fate of the teeth and their supporting tissues, after these structures have been involved in periodontal disease of varying degrees of severity. Such prediction depends upon a careful scientific evaluation of the sum total of factors which comprise known and unknown etiology, which determine the probability of eliminating the disease by means available to the periodontist, and which enhance the possibility of maintaining the status of a functionally dynamic reparation. These are the factors which constitute prognosis and indicate just what the probable course or outcome of disease would be.

Ordinarily, the prognosis of periodontal disease may be good, fair, hopeless or doubtful. It is to be expected that in such a general classification, there is apt to be some overlapping of the criteria of estimation. Much depends upon the training and experience of the clinician. Positive and negative prejudices would necessarily be instrumental in determining his own criteria, and as a result, what might constitute a fair prognosis in one case would not elicit a similar appraisal from another clinician of different background, training and experience. This state of affairs should not be too upsetting, however, and the scientific clinician will merit some legitimate satisfaction if he bases his estimation upon an attentively designed and wholly inclusive diagnosis. Therefore it would seem in order to review some and indicate other criteria which make prognoses good, fair, hopeless or doubtful, so that the clinician can be reminded constantly of those factors which need to be kept in mind when dealing with patients and their periodontal ills.

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1. PROGNOSIS GOOD

This indicates that there is a definite probability of eliminating the diseased condition. The supporting tissues can be restored to health with retention of the teeth for periods of their average life expectancy.

The objective organic factors which comprise a good prognosis may be conveniently classified into clinical and roentgenographic.

Clinically, there are the following criteria:

(a) *Color, tone and vitality of tissues.*

In addition to the diagnostic importance of color changes,[1] there is in the prognosis of periodontal disease an equally important role in which these changes participate. If by conservative means, the color, tone and vitality of the gingival tissues can be readily reactivated, then the prognosis may be good. Such cases are exemplified by the immediate response of acutely inflamed gingivae to the removal of the locally responsible factors.

(b) *Elimination of causative factors.*

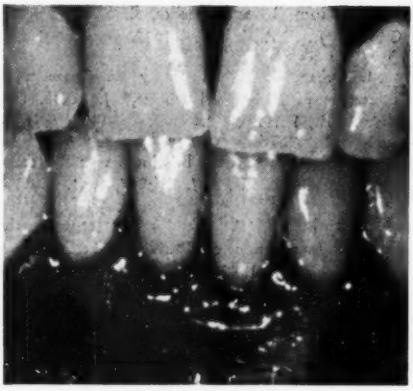
The definite probability of expelling the disease condition presupposes a precise recognition and possibility of removal of the etiologic factors of periodontal disease. In cases of incipient traumatogenic occlusion a favorable prognosis can be assured. The relationship between a known etiology and a good prognosis (Cases 1 and 2), is expressed by Miller[2] who indicates that "prognosis might be said to be favorable in proportion to the possibilities of complete removal or the etiological factors."

(c) *Mobility.*

In those cases in which there is an observable indication of movement, just slightly in excess of the normal, the prognosis is usually good. The causes of this mobility should be ascertained and correction instituted before there is an opportunity for increased movement, the results of a widening of the periodontal membrane space.

(d) *Extent and arrangement of reduced dentition.*

The larger the number of teeth remaining in the mouth, the better is the prognosis, since a larger number of teeth in the arch would be better able to withstand the stress and strain of masticatory forces. The arrangement of the remaining teeth is also an important factor in determining the prognosis. An arrangement which would allow for a more or less equalized dispensation of pressures, the latter so distributed in amount and direction as to elicit no immediate pernicious effects, would in turn contribute to the probability of retaining the teeth.



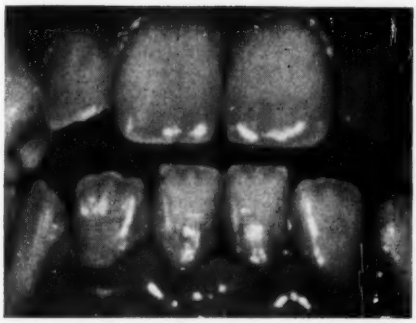
Case 1. Male. Age 40. Medical Diagnosis: None. Oral Diagnosis: Marginal Periodontitis originating from neglected gingivitis. Patient originally disinterested and uninformed about oral health. Prognosis: Good.

(e) *Relative depth and morphology of pathologic periodontal tissue detachment.*

The more superficial the detachment, the more favorable are the conditions at the gingival margin and the better is the prognosis. It is difficult to indicate exactly at what depth there would be a "normal" sulcus, because the depth of the sulcus, under normal conditions varies from zero to six millimeters, with an average of 1.8 mm.[3] Furthermore, if signs of pathology in the investing tissues are absent, then regardless of the depth, the sulcus may be termed "normal." In marginal periodontitis of local etiology, the detachments are shallow and the crest of the alveolus tends to appear somewhat resorbed in a horizontal direction. In these cases the prognosis is good.

Insofar as the pocket morphology is concerned, it can be indicated that the prognosis would be favorable as long as the bottom of the detachment can be easily reached through periodontol instrumentation. In other cases where the detachment is irregular and devious, it is impossible to reach certain cementum areas and the prognosis becomes uncertain.

The preceding clinical criteria are con-



Case. 2. Female. Age 26. Medical Diagnosis: None. Oral Diagnosis: Localized Gingival Hyperplasia resulting from local irritation. Patient cooperative. Prognosis: Good.

cerned specifically with oral considerations and are to be distinguished from the following systemic factors which are equally important.

(a) *Age.*

Certain forms of periodontal diseases have been generally regarded as conditions which occur in older individuals. These forms are mainly the degenerative periodontal pathoses. Acute periodontal lesions occur frequently in younger age groups. The chronic disorders are less frequently seen in children and adolescents. As a result, when degenerative periodontal conditions occur in young individuals, the prognosis is very much more unfavorable than when these conditions occur in older individuals. Miller[4] has pointed out that all things being equal, older patients with the same amount of bone destruction give better relative response to treatment than younger people, so the older the patient the better the expectations of complete response to treatment.

(b) *Degree of activity.*

Closely allied with the question of age is that of degree of activity, alertness and general interest of the patient in himself and in his environment. The greater the degree of activity, the greater may be his interest and ability to cooperate in carrying out those instructions which have been advised and which would be beneficial.

(c) *Health.*

Formerly emphasizing physical well-being, the concept of health now includes in addition, mental and social comfort. The absence of physical disease and infirmity are no longer the sole criteria of health.

Therefore, the total health of the patient, —his physical, mental and social adjustments—all of these things individually and together must, in greater and in lesser degrees, have some bearing upon the eventual outcome of the disease condition. If the adjustments are harmonious, the prognosis in all probability will be good.

Roentgenographically, there are the following criteria:

(a) *Quantity of alveolar support.*

The prognosis is good when the roentgenogram shows much bone tissue around the periodontally involved teeth and those teeth adjacent to the affected ones. Miller[5] and Goldman[6] indicate that the lesser the amount of remaining bone, the poorer the prognosis. At least one-half of the bone should remain around the root of a tooth to insure a good prognosis.

The absence of roentgenographic evidence of bone resorption and increase in width of the periodontal membrane space, do not preclude the possibility of tooth mobility.

(b) *Character of Bone.*

The prognosis is good, if the roentgenogram shows a dense even trabeculation with closely woven bone and small medullary spaces. A slight thinning of the lamina dura may indicate the beginning of periodontal disease. At this stage correct immediate treatment will insure a good prognosis.

(c) *Thinning of Alveolar Crest.*

This is one of the earliest signs of periodontal disease and the roentgenogram is actually the only means of detecting this early stage. Immediate elimination of causative factors will check the thinning process and assure a good prognosis.

2. PROGNOSIS FAIR

Cases with a fair prognosis are those in which it is felt that the periodontal tissues can most probably be restored to health with retention of the teeth for a reasonable portion of the periods of their average life expectancy. Here, there is good reason to believe that the disease condition can be eliminated.

The objective factors comprising a fair prognosis are based on the following clinical criteria:

(a) *Restoration of appearance and tone of gingival tissues.*

Case 3. Male. Age 45. Medical Diagnosis: Sinusitis. Oral Diagnosis: Periodontal Atrophy resulting from over-extended gingival restorations and improper tooth brushing. Patient interested and highly cooperative. Prognosis: Fair.

In cases of periodontal disease of long duration, it is usually the tardiness of diagnosis, or the degree of oral health negligence which may be responsible for destructive and irreparable changes, the consequences of chronicity. These changes are associated with poor tissue tone, and an unhealthy color of the gingivae. Recognition of etiologic factors and institution of correct treatment are usually enough to restore healthy color and tone, but restoration to the original form and contour of the gingival tissues are not as easily achieved. These considerations combine to make a fair prognosis. (Case 3)

(b) *Diagnosis and elimination of etiologic factors.*

In cases where the causative factors are recognized and defined but their removal is somewhat more involved, the prognosis is usually apt to be fair. The recognition of a deficiency in Ascorbic Acid with attendant effects upon the oral tissues is illustrative of a case in which the etiology of the condition may be defined. The treatment of such a case involves systemic therapeusis in addition to the local prophylactic procedures which must be instituted, hence these cases might be regarded as being relatively more difficult to treat, and also to diagnose, for that matter.

(c) *Stability of affected teeth.*

This symptom may have progressed to the point where there is a definitely discernible movement of the involved teeth. If stabilization and a reasonable rest period can be secured, the prognosis of the affected teeth is most likely to be fair.

(d) *Number and distribution of teeth.*

When there has been a drastic reduction in the number of teeth, but the remaining teeth have good alveolar support and are so distributed as to insure a widely equalized dispensation of masticatory forces in any replacement appliance, then the prognosis of the remaining teeth is fair. In such cases, a major portion of the decision as to the retention of teeth depends upon the discretion of the operator who must carefully weigh every factor before arriving at a conclusion.

(e) *Depth of pocket.*

The diagnosis is apt to be fair in cases where a clinical examination reveals (1) a pocket approximately 4-5 mm. or less in depth, (2) a pocket depth due more to gingival hyperplasia than to a detachment of the epithelial attachment, (3) an absence of suppuration, (4) an absence of excessive hemorrhage.

(f) *General factors.*

The general factors of age, degree of activity and health will be similar to those considered in a good prognosis.

Roentgenographically, there are the following criteria of a fair prognosis:

(a) There has not been horizontal alveolar resorption in excess of 50%, so that there remains an acceptable amount of bone around the roots of the involved teeth.

(b) There is no resorption in the bifurcations and trifurcations of the posterior teeth.

(c) There is dense, closely woven bone with small medullary spaces.

(d) There is a minimum evidence of changes in the periodontal membrane and lamina dura.

3. PROGNOSIS HOPELESS

When the prognosis of periodontal disease is declared hopeless, it is inferred that there is no possibility of eliminating the disease condition and the periodontal tissues cannot be restored to health. As a consequence, the teeth will not be retained for any acceptable period of time. Their life expectancy is zero, and immediate extirpation is indicated.

The objective factors comprising a hope-

less prognosis are divided into clinical and roentgenographic.

Clinically, the following factors are of importance:

(a) Excessive mobility of teeth which can be depressed in the sockets. (Case 4)

(b) Few teeth remaining in the dental arch. (Case 5)

(c) Remaining teeth so distributed that excessive stresses will be imposed on certain teeth if replacements are constructed. (Case 5)

(d) Irremediable physiologic inadequacy of the periodontium.

(e) Unlikeliness of removing known etiologic factors.

(f) Unknown etiologic factors in the presence of rampant destructive processes.

(g) Periodontal pockets in excess of 7 mm. with excessive hemorrhage and extensive suppuration.

(h) Haphazard wandering of teeth, as in periodontosis. (Case 6)

(i) Rapid increase in width of the interdental spaces.

(j) Loose, hypertrophied, gingival tissues around elongated, extruded, malaligned teeth. (Case 7)

(k) In those cases in which there is the possibility of focal infection, the prognosis of periodontally involved teeth is distinctly unfavorable. In cases of infectious arthritis, tuberculosis and syphilis, the retention of teeth may be injurious in that they may serve as foci of infection. In these cases the teeth might better be removed. As a general resistive factor, physical health may be a decisive distinction between success and failure. Generally speaking, the shortest and least physically harassing treatment should be the rule in cases where patients are in poor physical health. Most certainly the patient's systemic health is the important issue and merits a greater consideration than the retention or loss of teeth.

(l) The shortest and least mentally harassing treatment should also be the rule in cases where patients are in poor physical health, and most especially in cases where patients are in poor mental health.

Roentgenographically, the following factors are of importance:

(a) Extension of periodontal pocket to the apex of the tooth root. (Case 4)

(b) Involvement of bifurcations and trifurcations of the posterior teeth. (Case 4)

Case 4. Male. Age 28. Medical Diagnosis: Apparently normal healthy male. Oral Diagnosis: Periodontosis of idiopathic etiology. In this case all 4 first molars were similarly affected. One (1) has already been lost, while the others are extruded and extremely mobile. The remaining teeth are apparently free from degenerative processes. Patient cooperative. Prognosis of affected teeth: Hopeless:

(c) Less than 50% of supporting bone remaining around the roots.

(d) Large cancellae in the supporting bone.

(e) A darkening of the bone margin with deep penetration into the alveolus.

(f) Periapical involvement of teeth with periodontal disease.

(g) Close approximation of tooth roots with insufficient septal bone.

(h) Abnormal increase in width of the periodontal membrane.

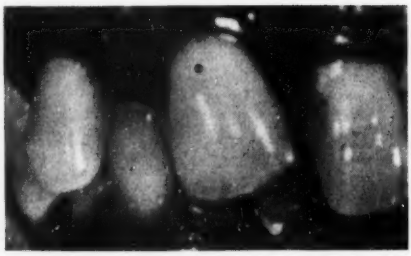
(i) Areas of tooth resorption.

(j) Excessive vertical bone resorption.

4. PROGNOSIS DOUBTFUL

A doubtful prognosis is one in which it is difficult to evaluate the probabilities of restoration to normalcy of the periodontal tissues and the retention of the teeth. In these cases it cannot be determined from a consideration of all factors on hand at the time of estimation whether or not the dis-

Case 5. Male Age 52. Medical Diagnosis: Arthritis and hypertensive cardiovascular disease. Oral Diagnosis: Oral Sepsis and severely depleted dentition with too few teeth remaining in the dental arch to render any replacements serviceable. Patient uncooperative. Prognosis: Hopeless, especially in view of medical diagnosis.



Case 6. Female. Age 36. Medical Diagnosis: Anemia. Oral Diagnosis: Generalized Periodontosis of nonspecific systemic etiology. Patient disinterested and uncooperative. In the case of migrating anterior teeth, prognosis is hopeless.

ease can be eliminated and therefore whether the prognosis will be fair or hopeless.

Among the factors which contribute to a doubtful prognosis are:

1. A lower molar bifurcation involvement, but with the tooth roots widely spread and well anchored in alveolar bone.

2. Radiographic evidence of a large amount of alveolar bone support, but clinical evidence of excessive mobility.

3. More than 50% destruction of bone around anterior teeth, without an accompanying excessive mobility.

4. Little possibility of completely eliminating known etiological factors.

5. Unknown etiological factors.

6. Presence of a chronic inflammatory condition which responds to conservative therapy but often recurs without provocation.

7. The general systemic condition such that no assistance can be expected in the matter of raising the general resistance and tone with benefit to all body tissues.

There are also many combinations of factors which make a prognosis questionable. The shift from a questionable to a fair or poor prognosis is dependent upon the accuracy with which the clinician is able to evaluate every factor presenting at the time the questionable prognosis becomes most imminent.

It is especially in cases with questionable prognoses that it becomes most necessary to evaluate critically the subjective constituents upon which all prognoses actually depend.

SUBJECTIVE FACTORS

Although much attention has been directed to the objective organic factors of prognosis in periodontal disease no similar amount has been devoted to the subjective factors which play an equally important part in prognosis and which are often decisive factors in making a questionable prognosis fair or a fair prognosis good.

Sympathetic understanding must always be given the factors which are determined to a large extent by a patient's personal characteristics. The level of intelligence and the particular emotional states which determine a person's capacity for mental activity are to be distinguished from the organic factors—clinical, roentgenographic and systemic—which have just been considered. The individual's capacities and emotional orientation embrace the subjective factors which must be appraised together with organic findings before rendering prognostic impressions or decisions.

A patient must understand the prescribed instructions for oral care before he can be cooperative. Inability to understand may result from a lack of education,

Case 7. Male. Age 58. Medical Diagnosis: Cerebral arteriosclerosis with psychotic reaction. Oral Diagnosis: Periodontitis complex resulting from oral sepsis, lack of oral care, unreplaced tooth loss, and generalized lowered resistance. Note the loose, hypertrophied gingival tissues around the malaligned, extruded teeth. Patient is very uncooperative. Prognosis: Hopeless.

Case 8. Male. Age 28. Medical Diagnosis: Schizophrenia. Oral Diagnosis: Chronic gingivitis resulting from the irritating effects of oral sepsis and a nonspecific factor inducive of tissue hyperaction. Patient is unreliable in that there are spontaneous periods of cooperation followed by periods of total apathy. Prognosis: Doubtful.

cultural deprivation, or from an inherent lack of mental ability. Usually the former two can be handled successfully with a favorable prognosis; however, a great deal of time and effort to make every step simple and concrete will be needed in the latter type of cases to achieve a modicum of cooperation.

Cases in which personality characteristics are involved constitute a much larger group and are much more difficult to handle. So often dental clinicians have found patients to be uncooperative and unwilling to follow the necessary directives for home treatment. (Case 8) It can be assumed that there are reasons why these people are uncooperative. If divulged by the patient, however, these reasons might not be regarded as being rational insofar as the dentist is concerned, but they are emotionally toned and are very real to the patient. There are fears and anxieties that may be peculiar to the particular individual. These are definite barriers to both pre-operative procedures and post-operative instructions. Quite often a kindly, considerate, middle-of-the-road attitude on the part of the dentist will make the patient divulge these fears and anxieties. Following this, an attitude of reassurance based upon a recognition of these factors even in the absence of intelligible causation, will usually change these persons from voluntary antagonists to relatively cooperative protagonists. One need not emphasize that cooperative patients are more likely to have favorable prognoses than those who give questionable cooperation. (Cases 1 and 2)

Much has been written about the necessity for the patient to be diligent in his home care of the oral cavity. Since the patient will have the responsibility for post-operative tissue care and maintenance, it is absolutely necessary to give him adequate instructions as to the proper procedures. Oral care is a time-consuming process and it is often difficult to get the patient to carry out all instructions which quite often include the continual usage of the toothbrush, the massaging of the gingivae, the interproximal stimulation, the overcoming of injurious incisal and masticatory habits and regular daily rinsing. These are among the many factors which can and do make the differences between successful, extended, or unsuccessful periodontal therapy.

There is a great need for periodontal treatment in patients who are mentally ill. Treatment in these cases may be attended with a larger number of difficulties. Consultation and cooperation with the psychiatrist in many cases will be the procedure of choice in achieving some patient cooperation and education. (Case 8)

The clinician's responsibility lies in ad-

Type or classification of Periodontal Disease. (After Orban)	*Etiology*	*Estimate of amount of dependence upon improvement of objective factors.*	*Estimate of amount of dependence upon absolute patient cooperation.*	*Prognosis of cases in which factors in columns 3 and 4 are operating jointly.*
I. *GINGIVITIS* (Inflammation)				
A. *Localized*				
1. Marginal	Known (Irritation)	90%	5%	Good
2. Hypertrophic	Known (Irritation)	90%	5%	Good
3. Chronic	Non-specific (Mild irritants and lowered resistance)	60%	10%	Doubtful
4. Acute	Known (Fusospirochetosis)	85%	10%	Good
5. Subacute	Non-specific (Infection and systemic resistance)	60%	10%	Doubtful
6. Edematous	Non-specific (Irritant and inherent tissue response factor)	55%	5%	Doubtful
7. Exudative	Known (Infection)	80%	5%	Fair
B. *Generalized*				
1. Marginal 2. Hypertrophic	Known in some cases (eg. Scorbutic and drug gingivitis)	50%	25%	Fair
3. Chronic 4. Acute 5. Idiopathic	Non-specific or unknown in other cases (eg. Endocrine and Idiopathic)	25%	5%	Poor
II. *PERIODONTITIS* (Inflammation)				
A. *Localized*				
1. Marginal	Known (Neglected gingivitis and irritation as in gingivitis)	80%	10%	Good
2. Chronic	Non-specific (Mild irritants and lowered resistance)	50%	10%	Doubtful
3. Acute	Known (Irritation and infection)	75%	5%	Fair
B. *Generalized*				
1. Marginal	Known (Scorbutic etc.)	80%	5%	Fair
2. Chronic	Known (Debilitating Diseases)	40%	10%	Doubtful
3. Secondary to Periodontosis	Non-specific	20%	5%	Poor

Type or classification of Periodontal Disease. (After Orban)	*Etiology*	*Estimate of amount of dependence upon improvement of objective factors.*	*Estimate of amount of dependence upon absolute patient cooperation.*	*Prognosis of cases in which factors in columns 3 and 4 are operating jointly.*
4. Systemic disturbances	Known but non-specific (Metabolic and endocrine disturbances)	40%	5%	Doubtful
5. Idiopathic	Unknown	20%	0%	Poor
III. *PERIODONTOSIS* (Degeneration)				
A. Localized	Known but non-specific	45%	25%	Doubtful
B. Generalized	Non-specific contributory factor	25%	5%	Poor
C. Idiopathic	Unknown	10%	0%	Poor
IV. *PERIODONTAL TRAUMATISM* (trauma)				
A. Localized	Known (Faulty dentistry, injurious habits, etc.)	70%	15%	Fair
B. Generalized	Known, but often non-specific (eg. functional insufficiency)	50%	5%	Doubtful
V. *PERIODONTAL ATROPHY* (atrophy)				
A. Localized	Known (eg. cross toothbrushing, injurious habits)	70%	15%	Fair
B. Generalized	Non - specific (Functional insufficiency)	40%	5%	Doubtful
C. Idiopathic	Unknown	20%	5%	Poor
VI. *GINGIVAL HYPERPLASIA* (Hyperplasia)				
A. Localized	Known (Irritants)	85%	5%	Good
B. Generalized	Known (eg. Drugs)	40%	35%	Fair
C. Idiopathic	Unknown	20%	5%	Poor

vising every patient who is being treated for periodontal disease of the importance of full cooperation if a good prognosis is to be assured. It is usually inadvisable to continue with conservative treatment if some cooperation of the patient cannot be obtained.

PROGNOSIS IN TYPE CLASSIFICATION OF PERIODONTAL DISEASE.

In the accompanying chart, there is listed in column 1 the particular types of periodontal disease. The types are based upon Orban's classification[7] of the pathology of

periodontal disease. From previous listings of the etiology of periodontal disease, a general statement is indicated in column 2 as to whether the etiologic factors are known, non-specific or unknown. In column 3 there is a percentage estimate of the expected clinical improvement in periodontal pathology which will occur if there is an elimination or amelioration of those objective organic factors, indicated as being responsible for the disease condition. This is more or less dependent upon therapeutic procedures instituted by the dentist himself. This evaluation will vary according to the background and experience of the clinician and will not approach a consistent degree of scientific accuracy until there has been established some universally acceptable index of periodontal disease. In column 4 is another percentage estimate of the expected improvement over and above that in column 3 which will accrue from absolute patient cooperation in carrying out given directions. In column 5 there is listed the prognosis arrived at as a result of a total appraisal of the indications in the preceding columns.

Upon the basis of an arbitrary scale, the different prognoses have been assigned. A total percentage of 90 and above constitutes a good prognosis, while percentages of 89 to 75 indicate a fair prognosis. From 74% to 41% the prognosis is doubtful and below 40% the prognosis is poor.

It will be seen that the most favorable prognoses occur in inflammation and hyperplasia, and the most unfavorable in degeneration. More specifically, it can be stated that in the localized forms of gingivitis, periodontitis and gingival hyperplasia, prognosis is best, while in generalized periodontosis, the prognosis is worst. Also, in the idiopathic forms of generalized gingivitis, generalized periodontitis, periodontosis, periodontal atrophy, and gingival hyperplasia, prognosis appears to be most unfavorable.

SUMMARY

1. Definition of prognosis in periodontal disease.
2. Objective organic factors comprising a good prognosis.
   (a) Clinical factors
   (b) Roentgenographic considerations
   (c) Systemic factors
      (1) Age
      (2) Degree of activity
      (3) Health
3. Objective factors comprising a fair prognosis.
   (a) Clinical
   (b) Roentgenographic
4. Objective factors comprising a hopeless prognosis.
   (a) Clinical
   (b) Roentgenographic
5. Factors contributing to a questionable or doubtful prognosis.
6. Subjective factors in prognosis of periodontal disease.
7. Prognosis in type classification of periodontal disease.

CONCLUSION

The preceding factors have been discussed in order to indicate to the clinician those criteria which determine the prognosis of periodontal disease. Special attention must be directed to the subjective factors which are often de-emphasized.

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Relation of Gingivitis to Dental Caries and Malocclusion in Children 14 to 17 Years of Age

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Chicago, Illinois

INTRODUCTION

DENTAL caries is often said to have a direct and causal relationship to gingivitis (Black, 1913; Coolidge, 1931; Fish, 1944; Shay and Smart, 1945). Malocclusions of the teeth have also been quoted as the basis for many types of gingivitis as well as periodontal destruction (Noyes, Schour and Noyes, 1938; Fish, 1944).

These statements are generally based upon clinical observations rather than quantitative data. The reason for this is probably the fact that quantitative and objective methods of assessing these three major dental disorders have become available only recently.

In 1938, Klein and Palmer initiated the extensive use of a quantitative method of evaluating dental caries in large groups (DMF Index[1]). More recently, Massler and Schour (1949) presented the P-M-A Index[2] for the quantitative assessment of gingivitis in large population groups. Massler and Frankel (1950) recently suggested a method for the quantitative assessment of malocclusion in large groups.

In view of the availability of these three methods of assessment, it was felt that a quantitative study of the relation of gingivitis to dental caries and to malocclusions could now be undertaken. It was hoped that such a study might serve to elucidate any relationships among the three major dental disorders which afflict man.

MATERIAL

This study was based on the examination of 2774 children of Morton High School, Cicero, Illinois. Practically 100 per cent of this school's population was examined so that no factor of selection might appear in the data. The children ranged in age from 14 to 17 years and included an almost equal distribution of males (1291) and females (1483). Age was considered in years from the last birthday.

The children examined came from a group of upper middle class families who lived in and near Cicero. The general health of the population group examined was very good—very little evidence of malnutrition being observed during the survey. The dental care of the children was considerably above the average in both quantity and quality. Gold restorations were common. Relatively few children required immediate dental attention. Many children were under active orthodontic treatment. These children were omitted from this study.

A teen-age group was selected for this study because this age level represents that period in life when growth and development of the dentition is newly matured. At this age the three most widespread oral afflictions, gingivitis, dental caries and malocclusion, are rarely complicated by general systemic disease or by aging. The gingivae and gingivitis are not complicated by degenerative processes which usually follow after 25 years of life. This age also experi-

1. DMF Index—decayed (D), missing (M) and filled (F) teeth or tooth surfaces.

2. P-M-A Index—papillary (P), marginal (M) and attached (A) gingiva.

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ences the highest attack rate by caries (Massler, 1945). Finally, physiologic malpositionings of the teeth so common between 6 to 12 years have usually become either self-corrected or permanently fixed by the 15th year (Broadbent, 1941). The evaluation of these three oral afflictions at the teen-age level is, therefore, less likely to be confused than at an earlier age (mixed dentitional period) when the dentition is in a state of flux, or at later ages when these processes have become complicated and obscured by other disorders and by aging.

METHODS

Method of Examination

The oral examinations were carried out by three separate teams, one each for gingivitis, caries and malocclusion. Each team consisted of two persons previously trained in the particular method of examination and in the criteria of assessment of the lesions to be examined. One person acted as examiner and the other as recorder. The examiner and recorder interchanged after every tenth examination in order to prevent fatigue and lessen subjective opinions or trends on the part of the examiner.

Methods of Assessment

Quantitative evaluation of the amount of gingivitis, dental caries and malocclusion present in each case examined was undertaken according to the following methods:

Gingivitis. The amount of gingivitis was assessed according to the method suggested by Massler, Schour and Chopra (1950). The number of papillae, margins and attached gingivae affected by inflammation (P-M-A) were counted and recorded according to the instructions outlined by Massler, Schour and Chopra (1950). The examination was conducted with a tongue blade. Only the buccal and labial surfaces were examined and recorded.

The data were recorded as three numerical values (e.g., P-M-A equals 2-1-0). Since such values do not lend themselves readily to simple statistical analysis, the three figures (P, M and A) were added together as P+M+A. The resulting value (P+M+A) was used in this study to denote the total number of gingival units affected. The validity of this procedure was tested by the rank correlation method, i.e., each value was used separately, to determine the ordinal rank of each case. Both methods of gingival evaluation gave identical results.

Dental Caries. The DMF index method as described by Klein and Palmer in 1938 was used in the assessment of dental caries experience. Examinations were made with No. 3 plain mouth mirrors and explorers.

Malocclusion. The number of malocclcluded teeth in each person was assessed according to the method suggested by Massler and Frankel (1950).

Each one of the three methods cited yield reliable numerical data which are susceptible to statistical analysis.

Grouping of Data

It was found necessary to group the data obtained into some system which would permit more effective use for comparisons and correlations and would also permit easier statistical evaluation. For this purpose frequency tables were first constructed from which five categories were obtained based upon the arithmetic mean for the group:

a) not affected—all zero values;
b) very mild (caries, gingivitis or malocclusion)—all values from one to half average;
c) mild—all values between one-half average to the arithmetic mean for the entire group;
d) moderate—all values from the average to twice the average;
e) severe—all values greater than twice the average for the group.

This system has the advantage of avoiding subjective designations to the terms mild, moderate and severe. Nevertheless, it

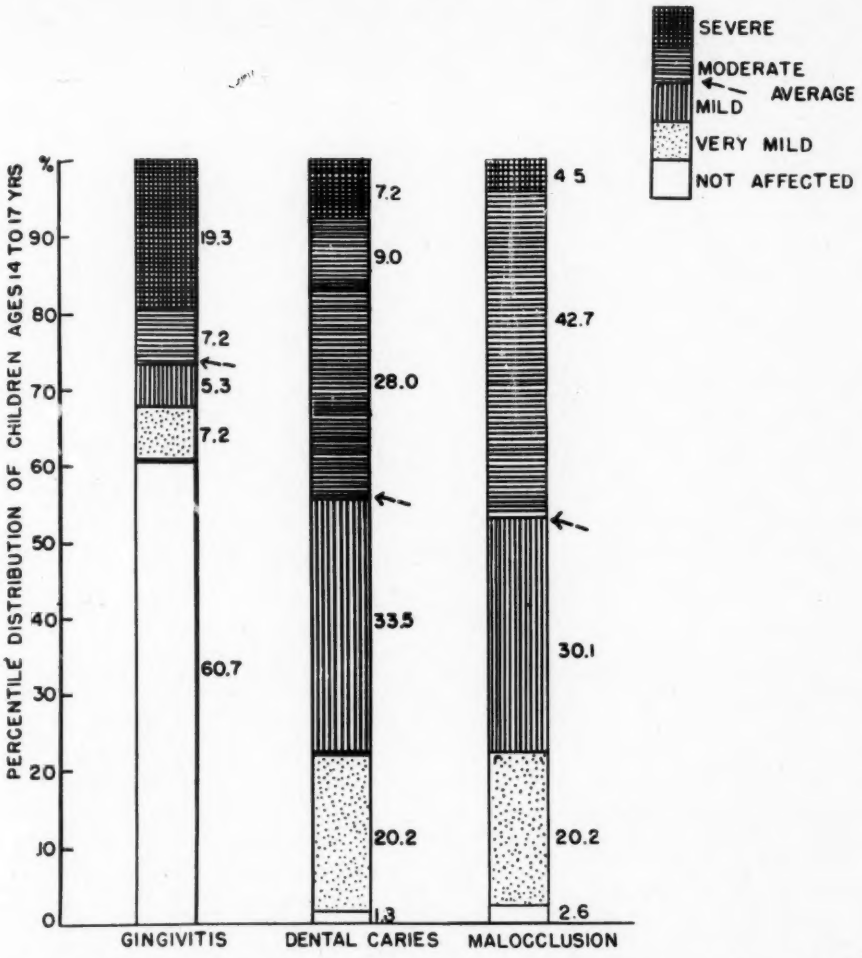

Fig. 1. Bar diagram showing the percentile distribution of children according to the severity of gingivitis, dental caries and malocclusion. Very mild, all values from one to half the average; mild, all values between one-half average to the arithmetic mean for the entire group; moderate, all values greater than the average to twice the average; severe, all values greater than twice the average for the group.

Note the "normal" distribution of persons with caries and malocclusion and the highly leptoskewed distribution of gingivitis experience.

permits a grouping of the cases into clinically observable categories and also permits simpler statistical evaluation (figure 1).

Statistical Methods. All data were tested for statistical significance using formulae suggested by Snedecor (1946).

FINDINGS

Relation of Gingivitis to Dental Caries

Scatter diagrams of the relation between the number of gingival units affected to the number of carious (DMF) teeth surfaces in each person at each age level from 14 to 17

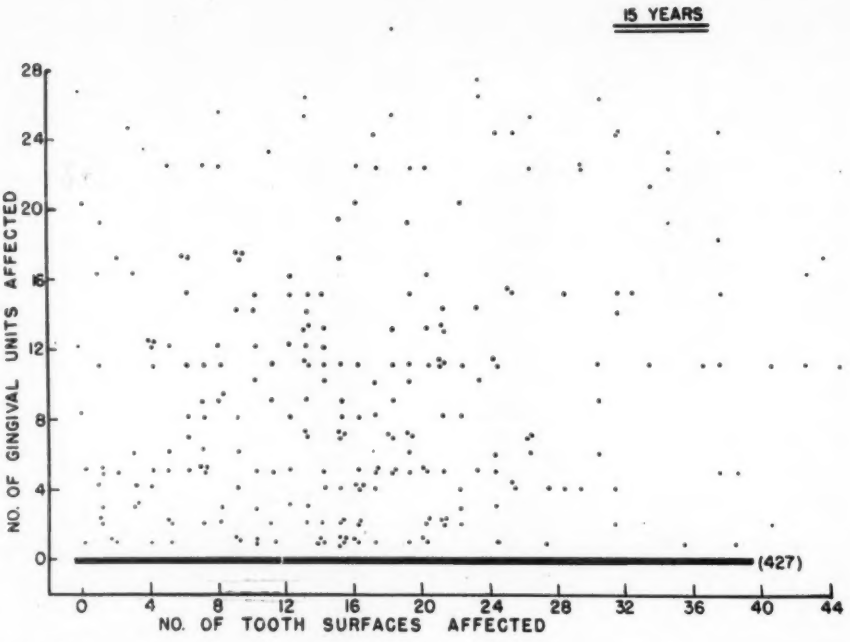

Fig. 2. Scatter diagram showing the relation between dental caries experience (number of DMF tooth surfaces) and gingivitis (number of P+M+A units affected) in 728 males and females 15 years of age. Note the absence of any definite correlative tendency. The diagrams were similar at each age level (14 to 17 years) and for each sex.

years showed a wide scatter of points and absence of any definite trend (figure 2). It was therefore concluded that gingivitis was not related (directly or inversely) to dental caries during this age period.

Comparison of Dental Caries Experience (DMF Teeth and DMF Tooth Surfaces) in Children with No Gingivitis and Children with Severe Gingivitis. To further determine whether dental caries experience was in any way related to gingivitis experience, a comparison was made of the caries attack rates in 1637 children with no gingivitis and 527 children with severe gingivitis (table 1 and figure 3). The average dental caries experience of the no gingivitis group (18.2 DMF tooth surfaces) was 3.1 DMF tooth surfaces less than in the severe gingivitis group (21.3 DMF tooth surfaces). A consistent difference in caries experience could also be discerned between the two groups at each age level (table 1 and figure 3).

The difference between the two groups was analyzed statistically and revealed that the probability of such a difference occurring by chance was less than one in 1000 (value of t equals 5.243). The difference at each age individual level was also analyzed statistically and found to be significant.

The fact that a real difference in caries experience was found between the no gingivitis and severe gingivitis group was surprising in view of the lack of correlation in the scatter diagrams (figure 2). A comparison of the dental caries experience of the no gingivitis and the severe gingivitis groups was therefore made with the line of regression for the total population (figure 3). This comparison was most revealing. It showed that although the dental caries experience of the severe gingivitis group was

TABLE 1.

Comparison of Dental Caries Experience (DMF Teeth and Tooth Surfaces) in 1637 Children with no Gingivitis and 527 Children with Severe Gingivitis (Ages 14 to 17 Years)

Age in Years	*No Gingivitis Group*			**Severe Gingivitis Group*		
	No. of Persons Examined	*Average No. of DMF Teeth per Person*	*Average No. of DMF Tooth Surfaces per Person*	*No. of Persons Examined*	*Average No. of DMF Teeth per Person*	*Average No. of DMF Tooth Surfaces per Person*
14	352	8.32	14.85	94	9.34	17.48
15	427	9.14	17.12	151	10.31	20.29
16	379	10.13	19.62	132	11.27	22.35
17	479	10.98	20.50	150	12.18	24.04
14 to 17 Yrs.	1637	9.73	18.20	527	10.91	21.37

*More than nine P+M+A gingival units per person.

The average number of DMF Tooth Surfaces per person was significantly different in the No Gingivitis and Severe Gingivitis Group ($t=5.243$; $P=$ less than 0.001). This difference was also significant in males ($t=2.027$; $P=0.05$) as well as in females ($t=4.388$; $P=$ less than 0.001). The difference in females was especially marked.

significantly higher than that of the rest of the population, the caries experience of the no gingivitis group was *not lower* than that of the total group, but coincided with it exactly. This indicated that while the caries experience of the severe gingivitis group did differ from the rest of the population, caries experience was *not* correlated with gingivitis in any of the other groups with zero, very mild, mild and moderate gingivitis. This explained the significant differences in the means between the severe gingivitis group and the no gingivitis group since the latter represented also the rest of the population.

Comparison of Gingivitis (P+M+A) in Children with Low and High Dental Caries Experience. To further explore the relationship of gingivitis to dental caries, the gingivitis experiences of the low and high dental caries groups were compared (table 2). This analysis showed that children in the low caries group had an average of 3.5 gingival units affected, while children in the severe caries group showed 6.2 gingival units af-

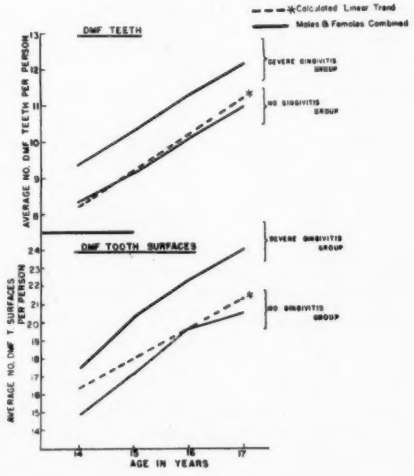

Fig. 3. Graph comparing the dental caries experience (DMF teeth and DMF tooth surfaces) in 1637 children with no gingivitis and 527 children with severe gingivitis (ages 14 to 17 years, males and females combined). The difference in the caries experience between the two groups is statistically significant ($t=5.243$).

TABLE 2.

Comparison of Gingivitis Experience (P+M+A) in 599 Children with Low Caries Experience and 443 Children with High Caries Experience (Ages 14 to 17 Years).

Age in Years	Low Caries Group (0 to 9 DMF Tooth Surfaces Affected)		Severe Caries Group (More than 31 DMF Tooth Surfaces Affected)	
	Number of Persons Examined	Number of Gingival Units Affected (P+M+A) per Person	Number of Persons Examined	Number of Gingival Units Affected (P+M+A) per Person
14	148	2.74	41	7.6
15	181	4.10	92	7.14
16	140	3.69	135	5.89
17	130	3.63	175	5.59
TOTAL 14 to 17 Yrs.	599	3.57	443	6.20

The average number of gingival units affected (P+M+A) per person was significantly different between the low and severe caries groups (t=5.968; P was less than 0.001). This difference was also significant in females (t=6.942; P=less than 0.001) but weak in males (t=2.633; P was 0.01). The differences were also tested at each age level.

fected (table 2). This difference in gingivitis experience between the two groups was statistically significant (t equals 5.968: p equals 0.001). Further comparison of the gingivitis experience of the two groups showed that a difference was present at each age level and that all of these differences were significant (table 2). However, a sex difference was present. The difference in gingivitis experience between children with a high and low caries experience was statistically significant for females alone, for females and males combined, but *not for males alone* (figure 4).

In view of this difference in gingivitis experience between the two groups, the data from both were plotted against the line of regression representing the total group. This analysis revealed again that the high caries group had a higher gingivitis experience than did the other groups but that the gingivitis experience of the low caries group coincided with that of the total group. Again this points to the fact that *while children with a high caries experience tend to have severe gingivitis, no correlation between caries and gingivitis experience existed in the rest of the children.*

Relation of Gingivitis to Malocclusion

Scatter diagrams showing the relation between the number of gingival units affected and the number of malocclusion teeth in each person at each age level indicated that no correlation existed between the two conditions during this age period (figure 5).

Malocclusion Experience in Children with No Gingivitis and Children with Severe Gingivitis. In order to further explore the question of any possible relation between gingivitis and malocclusions, the average number of malocclusion teeth was calculated for 1483 children with no gingivitis and 459 children with severe gingivitis. The average number of malocclusion teeth in the former was found to be 10.0 while the number in the latter group was 12.26. A difference was consistently present at each age level. This difference when analyzed statistically was found to be sig-

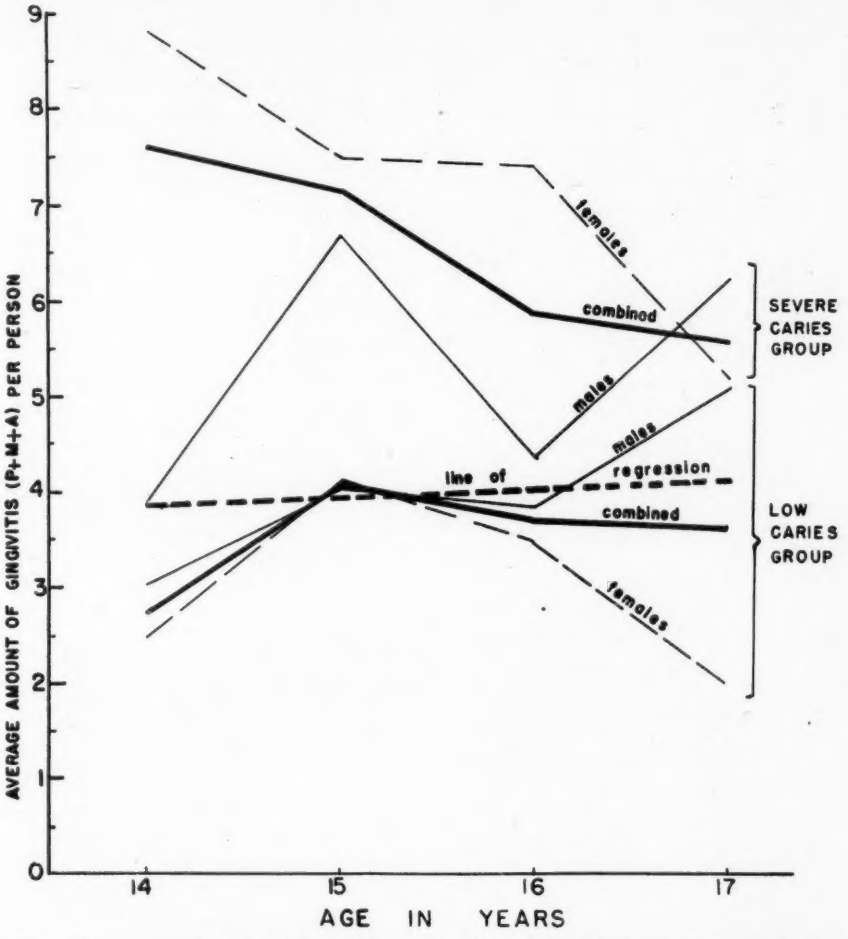

Fig. 4. Graph comparing gingivitis experience (P+M+A) in 599 children with low caries and 443 children with high caries attack rate. The difference in gingivitis experience between the two groups is statistically significant for females alone ($t=5.968$), females and males combined ($t=6.942$), but not for the males ($t=2.633$). The line of regression represents the linear trend for the entire group.

nificant (t equals 7.189, p is less than 0.001). Analysis of the data for males and females separately further substantiated the fact that an actual difference in the number of maloccluded teeth existed between children with no gingivitis and children with severe gingivitis (table 3).

In this analysis, and as in the previous section, the phenomenon of a significant difference in the number of maloccluded teeth between children in the no gingivitis and severe gingivitis groups where earlier findings revealed no correlation (see figure 5), required some explanation. The data for the no gingivitis and the severe gingivitis groups were therefore compared to the line of regression representing the incidence of malocclusion for the entire group of children (figure 6). This analysis revealed that

TABLE 3.

Comparison of the Average Number of Malocclued Teeth in 1483 Children with No Gingivitis and 459 Children with Severe Gingivitis (Ages 14 to 17 Years).

Age in Years	No Gingivitis Group		Severe Gingivitis Group*	
	Number of Persons Examined	Average Number of Malocclued Teeth per Person	Number of Persons Examined	Average Number of Malocclued Teeth per Person
14	323	9.79	81	12.92
15	410	9.87	141	11.88
16	356	9.56	118	12.39
17	394	10.7	119	12.11
14 to 17 Yrs.	1483	9.99	459	12.26

*More than nine gingival units (P+M+A) affected per person.

The average number of malocclued teeth per person was significantly different in the no gingivitis and severe gingivitis group ($t=7.189$; P=less than 0.001). The difference was also significant in males only ($t=3.697$; P=0.001) and females only ($t=6.261$; P=less than 0.001). This difference was statistically significant at each age level.

the number of malocclued teeth in the severe gingivitis group was much higher than that of the total population whereas, the number of malocclued teeth in the no gingivitis group was slightly less and overlapped that of the total population. We were therefore forced to the conclusion that while, in general, no direct correlation existed between the number of malocclued teeth and the amount of gingivitis, children with a high gingivitis experience tended to have a significantly higher number of malocclued teeth.

DISCUSSION

This study supports previous findings by Brucker (1943), Day (1944) and King (1945) that in school children and in young adults, gingivitis and dental caries are not related (either directly or inversely). No relation could be found between gingivitis and the number of malocclued teeth.

The date revealed, however, that children with severe gingivitis tended to have a higher caries attack rate and a greater num-

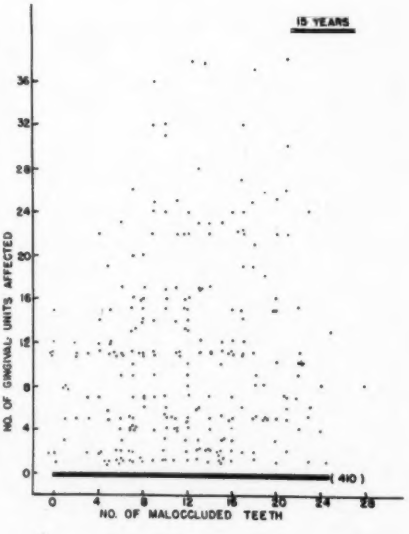

Fig. 5. Scatter diagram showing the relation between the number of malocclued teeth and the number of gingival units affected (P+M+A) per person in 728 children 15 years of age. Note the absence of correlative trend. The diagrams were similar at each age level (14 to 17 years) and for each sex.

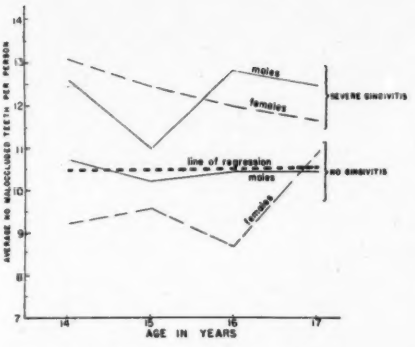

Fig. 6. Graph comparing the average number of malocclueded teeth in 1483 children with no gingivitis and 459 children with severe gingivitis. Note that the children with severe gingivitis have a significantly greater number of malocclueded teeth than the "average," but that the children with no gingivitis tend to show the same number of malocclueded teeth as does the average child.

ber of malocclueded teeth. Since this relationship was not found to occur in the rest of the population, it was concluded that the "severe" group constituted a separate and distinct group which merits further analysis. It is possible that children with a high caries attack rate might have a large number of open, unfilled cavities and a larger number of poor fillings. Such irritant factors might explain the high gingivitis experience, (Black, 1913; Shay and Smart, 1945). Similarly, it is possible that children with many malocclueded teeth tend to have many areas of food impaction and therefore more gingivitis (Black, 1913).

These findings in no sense deny the oft repeated, but still unproved, thesis that caries and periodontosis are mutually antagonistic disorders of the human mouth. Periodontal destruction is characteristic of a much older age group and represents etiologically and pathologically a different form of disorder than does gingivitis. This study only emphasizes the fact that a similar analysis of the relation of caries to other periodontal disorders should be initiated in older age groups.

SUMMARY AND CONCLUSIONS

This study was intiated in order to determine the relation of gingivitis to dental caries and malocclusion in 2774 children 14 to 17 years of age.

Quantitative methods of assessment were used for the evaluation of gingivitis (the P-M-A index as suggested by Schour and Massler, 1949) for dental caries (the DMF index as suggested by Klein and Palmer, 1938) and for malocclusion (the index of malocclusion as suggested by Massler and Frankel, 1950).

The data revealed that:

1. No relation existed between the number of carious teeth (DMF teeth or DMF tooth surfaces) and the number of inflamed gingival units (P+M+A) in this age group.

2. No relation existed between the number of malocclueded teeth and the amount of gingivitis (P+M+A).

3. However, children with *severe* gingivitis had a tendency to experience a higher caries attack rate as well as a larger number of malocclueded teeth than did the rest of the children.

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Calcified Bodies in the Gingiva*

By WILLIAM H. HIATT, D.D.S., *Denver, Colorado*

BIOPSY specimens of the gingiva taken from normal as well as pathological cases, revealed different types of "inclusion bodies."[1] These inclusions were of different types, such as cementum fragments, cementicles, calcified hyalin connective tissue, calcified thrombi and crystalline foreign bodies.

In an investigation of "the gingival structures in diabetes mellitus,"[2] similar calcified bodies were found in a large number of cases. In thirty-six biopsies taken from diabetics, ten calcified bodies were found. In comparison, forty biopsies of non-diabetics showed seven calcified bodies. It was felt that "since foreign bodies were found in more than one-fifth of our cases, there should be further study of their prevalence in a larger number of biopsies." Such a study on a larger material was undertaken and the result is reported herewith.

One-hundred-ninety biopsy specimens from that many individuals were examined from clinically "normal" cases.[3] The patients' ages ranged between ten years and seventy-nine years. Forty-eight calcified bodies were found in these one-hundred-ninety specimens which is 25.26%, or

(*) From the Colorado Dental Foundation, Colorado Springs, Colorado.

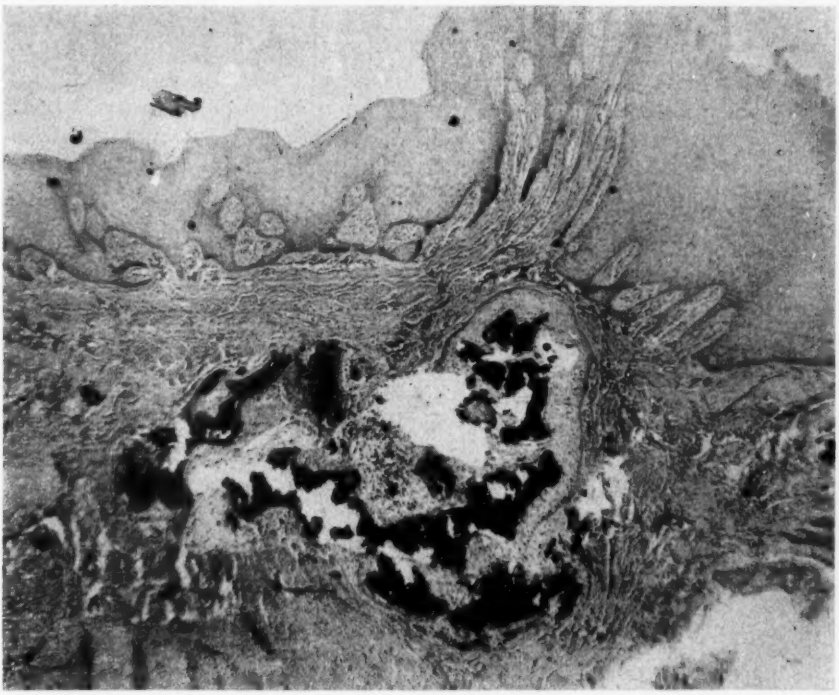

Fig. 1. Large calcified body in gingiva. Epithelium envelopes calcified tissue.

approximately one-fourth of the specimens. No significant difference in the number of calcified bodies was found in the different age groups. The variation is probably due to the number of cases, rather than the age differences.

Age	*No. of cases*	*No. of calc. bodies*	*Percentage*
10–19	14	5	35.71
20–29	38	7	18.42
30–39	41	12	29.26
40–49	55	12	21.81
50–59	12	3	25.0
60–69	15	5	33.33
70–79	4	1	25.0
unknown	11	3	27.27
Total	190	48	25.26

CHART 1

Chart *Number 1* shows the age distribution and number of cases. Most of the calcified bodies were of the degenerating and calcifying collagen — (Chart II); some originated probably in the periodontal membrane as cementicles; some were cementum remnants; and a few were of the phebolith type, shown also recently by Mikola and Bauer.[4]

The finding of such a number of calcified

Type of calcified bodies

Calcified connective tissue	36	
Cementicles	8	
Cementum remnants	2	
Phleboliths	2	

CHART 2

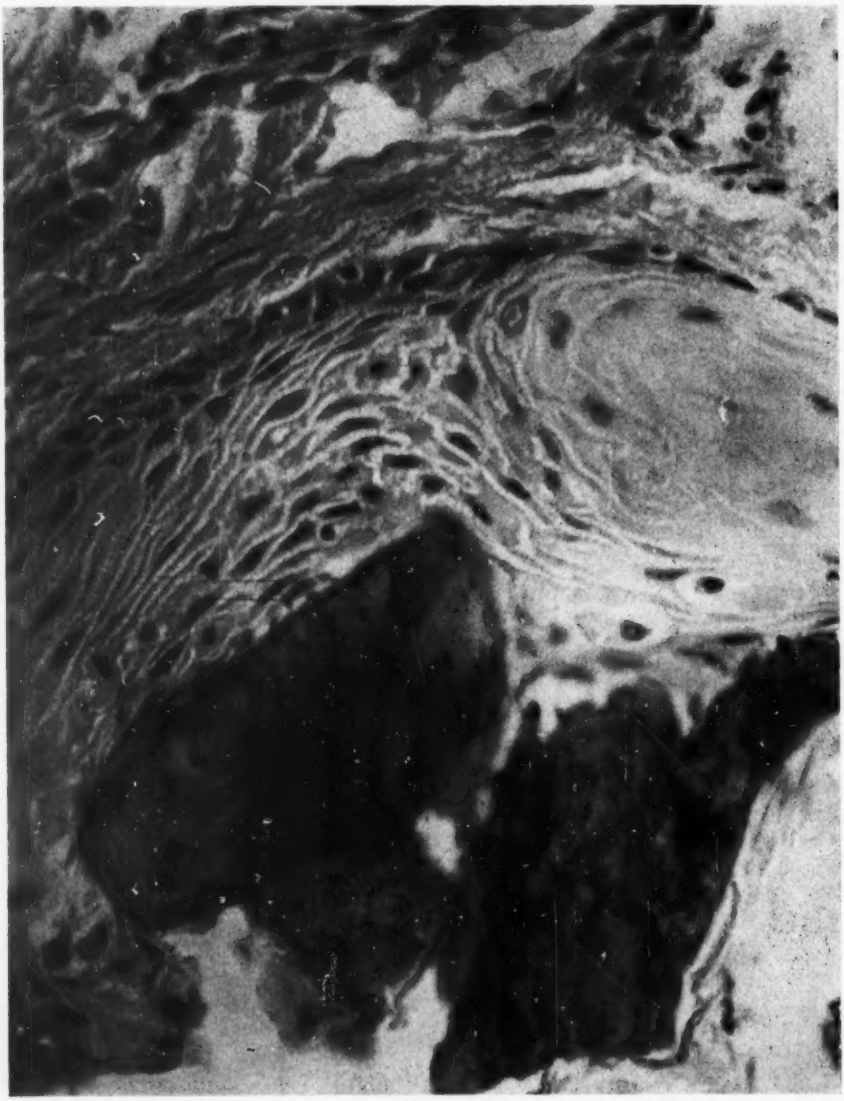

Fig. 2. Higher magnification of part of calcified body (Figure 1.), with stratified squamous epithelium surrounding calcified mass.

bodies in the gingiva might be interpreted to be the result of various irritations to which the gingiva is exposed. Such irritations may be from extrinsic factors which are physical or chemical in nature, or from intrinsic factors such as diabetes mellitus or other systemic disturbances leading to degeneration of connective tissue.

Figure 1 shows one of the largest calcified

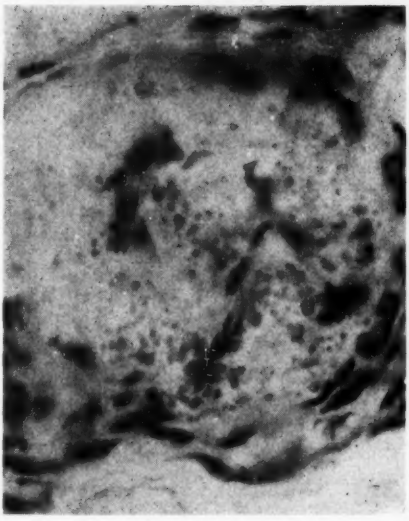

Fig. 3. Hyalin connective tissue with calcium droplets, indicating beginning calcification.

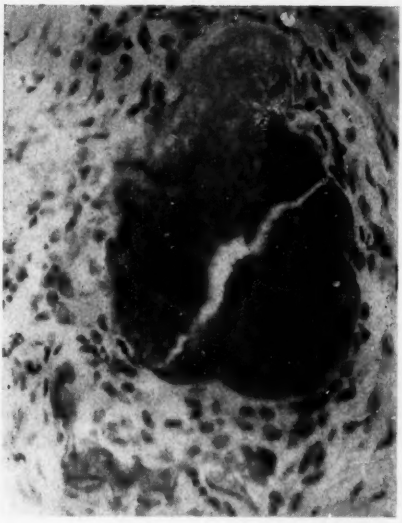

Fig. 4. Calcified body with granular appearance of calcification.

bodies found. It is of the calcified connective tissue type. In certain areas, an uncalcified, hard substance lines the surface of a calcified mass. In other regions, osteoclasts are resorbing the calcified structure. A large part of the calcified mass is enveloped by stratified squamous epithelium, which has proliferated from the oral epithelium as shown in higher magnification in *Figure 2*. Surrounding this calcified structure, and in areas where connective tissue is seen between calcified areas, inflammatory cells (plasma cells) are present.

It has been assumed that degenerating or necrotic connective tissue forms the base of many of these calcified bodies. One specimen (*Figure 3*), shows this very clearly. A hyalin connective tissue is seen with droplets of calcified material dispersed in it, indicating progress of calcification. In another specimen (*Figure 4*), the calcification has progressed to considerable extent, but the granular appearance still indicates the mode of development. The crack in the center of the calcified body has been found before in such calcifications.[1] Near the periphery of this calcified body, small pieces of calcified tissue are seen, some with empty bone lacunae and with osteoclastic resorption, leaving them with ragged edges.

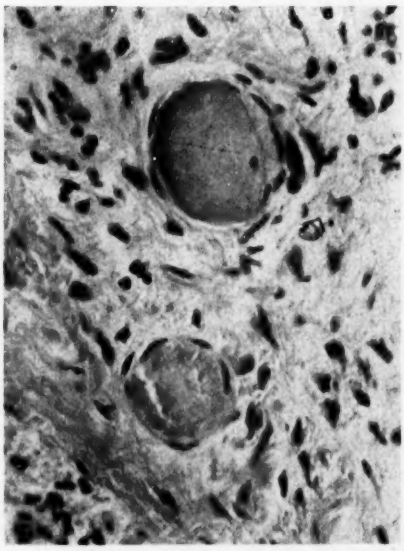

Fig. 5. Phleboliths (calcified thrombi in venules)

In *Figure 5*, two calcified bodies are seen, surrounded by flat cells, probably endothelial cells, and these can be considered phlebolites, calcified thrombosed venules. These have been described in literature previously, and are probably more frequent than formerly believed.

This statistical survey shows actually the minimum number of calcified bodies in the gingiva. In most of these biopsies only a single papilla was examined from each individual. In no instance was there any clinical sign of a calcified body within the papilla. If entire full-mouth gingivectomies had been examined, we can assume that more calcified bodies would have been found. We also have to take into consideration that no serial sections have been made. Only five to ten sections were examined from one biopsy specimen, and the calcified body often was found in two or three sections only, and not in the rest. Had serial sections been made from each biopsy, we can assume that the number of calcified bodies would also have been higher.

Summary. Calcified bodies (inclusion bodies) are a very frequent finding in the gingiva. In one-hundred-ninety specimens, forty-eight calcified bodies were found, or 25.26%. This percentage constitutes a minimal number of calcified bodies in the gingiva. The calcified bodies are of different character and origin: calcified hyalinized connective tissue, cementicles, phlebolites.

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(3) 140 specimens were from the material collected by Drs. A. W. Maier, F. M. Wentz and B. Orban at the University of Illinois and Dunnings Hospital for their study of age changes in the gingiva—(to be reported). The remaining specimens were obtained at the University of Denver from students and from biopsies submitted to the Histopathology Study Club of the Colorado Dental Foundation.

(4) Mikola, O., and Bauer, William: "Cementicles" and Fragments of Cementum in the Periodontal Membrane. J. Oral Surg., Oral Med. and Oral Path. **10**:63, 1949 (August).

The Colorado Dental Foundation

The Colorado Dental Foundation announces its Fifth Annual Seminar on Dental Medicine, which will be held on June 17 - 21, 1951, at the Fine Arts Center, Colorado Springs, Colorado.

Designed around the general theme, "Dental Medicine," the Fifth Seminar will present the following eminent essayists: Doctors Hermann Becks, Isaac Schour, Harry Sicher, J. L. T. Appleton, William A. H. Rettberg and Tom Spies.

Attendance is open to any member of the American Dental Association. The fee is two hundred dollars. Other courses offered by the Colorado Dental Foundation are:

Histopathology of the Supporting Tissues
June 11-16, 1951 ..... Fee $400.00

Theory and Practice of Periodontia
June 25-30 ..... Fee $200.00

These courses have been approved by the Veteran's Administration.

Case Report

Periodontosis in a Child Resulting in Exfoliation of the Teeth

By S. LEONARD ROSENTHAL,* D.D.S., *Philadelphia, Pa.*

HISTORY

C. F., female, 8 years old, complained of loose teeth, pain during mastication and a scaly appearance of the hands and feet.

Her parents, born in Spain, are apparently normal individuals with no dermatologic lesions and no unusual oral symptoms. The mother became edentulous in her fortieth year as a result of periodontal disease. The father exhibits filled, carious and missing teeth as would be expected of one who is indifferent to oral hygiene and good dental care. They could recall nothing of significance in the medical or dental history of their parents, brothers or sisters.

Four children were born to them after their migration to the United States. The first is a healthy and apparently normal man. The third, a young woman of 18, has enjoyed excellent health. Her skin is normal; her teeth are free from caries; and the gingivae are of excellent color and texture. One maxillary lateral incisor is congenitally missing.

The second child, a boy, developed a scaly appearance of the hands and feet during infancy which has persisted, despite treatment, throughout his 23 years of life. The deciduous teeth were lost early. When 11 years old, he was examined at Jefferson Hospital, Philadelpia, because several permanent teeth had been exfoliated and others were loose.

Radiograph of skull and teeth of boy: Examination of the skull is negative for evidence of increased intra-cranial pressure or bony erosion. The sella is normal in size and contour. There is an intra-cranial mottled opacity, the appearance and location of which is highly suggestive of calcification of the choroid plexus of the right lateral ventricle. The pineal is not calcified.

Examination of the teeth reveals the presence of rather sharply localized symmetrical areas of alveolar absorption which particularly involve the upper central incisors and upper bicuspids. There is a rather marked degree of horizontal alveolar absorption considering the age of the patient. The roots of the lower second and third molars are not visualized.

Radiograph of long bones: The bones, joints and epiphysis of the left and right wrist, elbow, foot and knee have a normal developmental appearance excepting the epiphysis of the left proximal phalanx of the left foot. This epiphysis is in two parts with a line of separation through its center running in the anterior-posterior diameter.

Laboratory studies Hb. 101; R.B.C. 5,000,000; W.B.C. 10,000; C.I. 1.01. Wass. negative, Kahn negative. Blood cholesterol—110 mgs.; blood calcium 9.77 mgs.; Blood phosphorus 4.45 mgs.; Blood Calcium—9.03 mgs.; Blood phosphorus—4.5 mgs.; Phosphatase 7.4; Blood cevitamic acid 2.8 mgs. Urinalysis: color—tur. amber; reaction—acid; albumin two plus; sugar—neg., crystals—0; blood—neg.; pus cells—occ.; casts—occ.; amorph. 0; epith. few.

The last teeth were removed and full dentures were inserted during the patient's sixteenth year.

MEDICAL HISTORY

The fourth child, the patient under study, was delivered without incident. A scaly appearance of the palms of the hands

Presented to the thirty-fourth annual meeting of the American Academy of Periodontology at Atlantic City, October 27, 1950.

*Assistant Professor of Periodontia, School of Dentistry, Temple University.

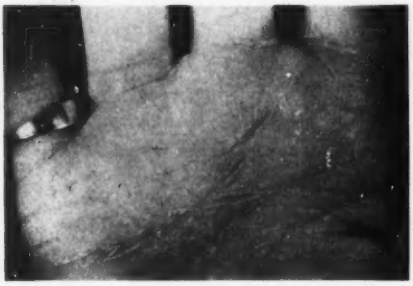

Figure 1

and soles of the feet was noted in early infancy. The deciduous teeth began to exfoliate at 22 months. The common childhood diseases were experienced but not in severe form. The permanent teeth erupted at the usual time but became mobile shortly after eruption.

EXAMINATION

A mentally and physically well-developed girl, eight years old, had painless cracks and exfoliation of the palmar and plantar surfaces (Fig. 1). The remaining skin and hair were normal. The first molars, cuspids and incisors had erupted. The first bicuspids had begun to appear. Roentgenographic examination revealed the first molar and incisor roots to be completely formed and the presence of all teeth except the third molars (Fig. 2). There was extensive alveolar resorption in both jaws.

The gingivae surrounding each tooth

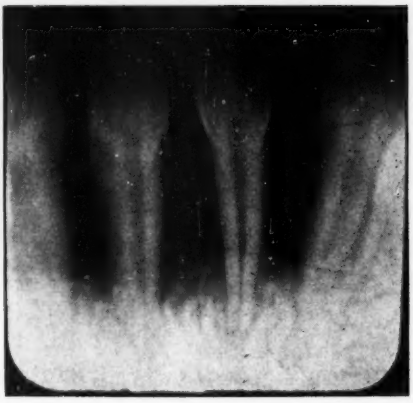

Figure 2 (A)

were hyperplastic, fiery red and edematous (Fig. 3). Where the teeth had not erupted, the gingivae were normal in color and consistency. Circumferential pockets averaged 6-8 mm. in depth. The buccal mucosa and other structures were normal.

The teeth had a 2-3 mm. mobility, were spaced, malposed and had large deposits of calculus.

Laboratory examination at Mt. Sinai Hospital of Philadelphia: Hb. 82% 11.8 gr., W.B.C. 10,300, Non seg. 4, Seg 41, Eos. 4, Lymph 46, Mono 5, Bl. Sugar 118, B.U.N. 8.5. Urinalysis: color—amber; reaction—acid sp. gr. 1.016. Miscell.—epith. mucus. Wass. (Kolmer) neg. Kline—neg. Vitamin

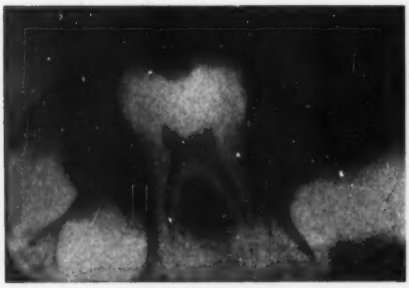

Figure 2 (B)

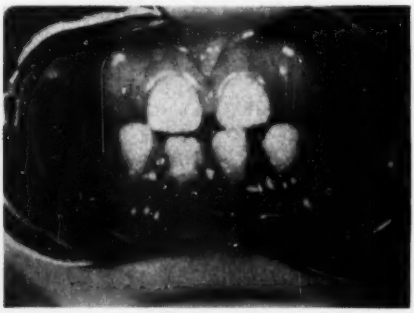

Figure 3

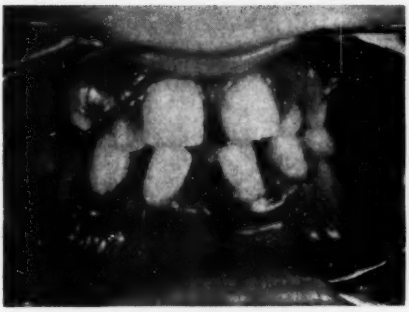

Figure 4

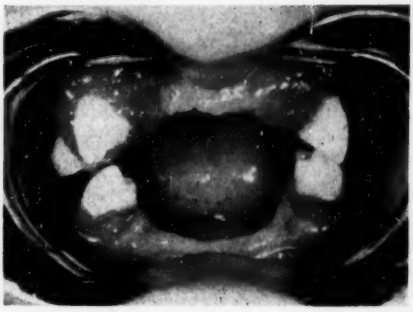

Figure 5

C 0.7 mg./100 cc. plasma; Phosphorus (serum) 3.1 mgs. per 100 cc. of blood. Phosphatase (alk.) 4.5 units. Blood sugar —112; Total Cholesterol—107; Calcium (serum)—12.2; Phosphorus (serum)—3.5; Blood culture 24 hrs.—no growth; Blood culture 48 hrs.—no growth. Subsequent blood counts were within normal limits.

X-ray of long bones: There are no osseous, periosteal or joint abnormalities present. Epiphyseal development is progressing normally.

X-ray of skull: The skull is normal in size, shape and density. There is no evidence of increased intracranial pressure. The Sella Tursica is normal in size and contour. The slinoid processes and the dorsum cellae are normal. The body of the sphenoid is only partially pneumatized.

TREATMENT

The deposits were removed from the teeth and proper home care was instituted. Although the diet was satisfactory, it was fortified with vitamins and daily injections of vitamin A were given for a month. The patient was kept under observation with periodic prophylaxis. The teeth continued to migrate and to loosen (Fig. 4). A year later, the maxillary right first molar was so loose that mastication was extremely painful. It was removed together with the adjacent tissues and sections were examined by Dr. Paul E. Boyle. He reported: "The epithelium is without cornification, is edematous and heavily infiltrated with polymorphonuclear leucocytes. The submucosa shows subacute inflammation. The endothelial cells of the vessels are greatly swollen. Plasma cells, lymphocytes and poly-

Figure 6 (A)

Figure 6 (B)

morphonuclear leucocytes are abundant. Eosinophiles are fairly numerous. Vacuolated macrophages (foam cells) are found. There are small areas of hemorrhage and of necrosis.

"The pulp shows dilated and congested blood vessels. The odontoblasts are well preserved, but in some localized areas they are obscured by finely granular eosin staining precipitate. In other locations this precipitate is found just pulpward to the odontoblasts.

"The periodontal tissues are almost completely lacking. The cementum is covered with filamentous organisms, some of which appear to be colonies of actinomyces. A purulent exudate adheres to the surface in places.

"Diagnosis: Acute and chronic inflammation of the periodontal tissues. Absence of cornification of epithelium."

Four years later (she was now twelve years old), multiple extractions became necessary and a partial denture was constructed at Temple University Dental School. There were no post-operative sequellae. The healed tissues were normal in appearance (Fig. 5, 6). The remaining teeth will be lost at an early date.

CONCLUSIONS

The dermal lesions were diagnosed as keratoderma plantaris et palmaris, a congenital ectodermal defect. It is reasonable to assume that this defect affected the oral tissue also. Certainly, the teeth were exfoliated as though they were foreign bodies. Once these irritants were removed, the tissues healed normally.

Editorials

Undergraduate Instruction in Periodontology

The purpose of dental education is obviously that of preparing the student to meet the dental health needs of the public. And foremost among these needs is the prevention and treatment of diseases affecting the supporting tissues of the teeth—the largest single cause of tooth loss in adult life.

This being true, it is not unreasonable to assume that undergraduate instruction in this field would be given a prominent place in the dental curriculum. Unfortunately this is not the case. Instead, it will be found to occupy little more than two percent as compared with twenty-two percent in reparative treatment. As might be expected, students are being graduated from our dental schools not only with little knowledge and less interest in periodontology, but what is more discouraging, with the impression gained in their student days that periodontia is of secondary importance in the practice of dentistry.

The inconsistency in this situation is forcefully set forth in two articles appearing in the January 1951 issue of the Journal of Dental Education by Harold J. Leonard[1] and Irving Glickman.[2]

[1]Leonard, Harold J. The Status of Periodontology in the Undergraduate Curriculum. J. of Dent. Ed. 15:97 (1951)

[2]Glickman, I. A Change in the Aims of Dental Education. J. of Dent. Ed. 15:109 (1951)

Glickman refers to the situation as "an inexplicable paradox—highlighted by an inconsistency between the nature of the dental problem of the population and the teaching program designed to prepare dentists to cope with this problem. On the one hand, the observer learns that beginning with the fourth decade of life, periodontal disease is the greatest single cause of tooth loss. In addition, because of a steadily increasing life expectancy, each year finds a large proportion of the population at an age level at which tooth loss is more likely to result from periodontal disease than caries—the provisions for periodontal training constitutes the Achilles heel of the undergraduate dental curriculum. It is a most vulnerable inadequacy which unfortunately annually bears a fruit of hundreds of improperly oriented dental school graduates whose most formative years of dental practice are spent oblivious to their insufficiency in the periodontal field."

With a view to correcting this situation Glickman recommended among other things the "crystallization of the interrelationship of clinical periodontology with the other branches of dentistry and inculcating the periodontal viewpoint into the thinking and overall practice of the student—convincing him that periodontal care is a health service which is a basic part of the general practice of dentistry, to be administered at all times it is needed and that no patient is completely cared for if his periodontal needs go unattended."

The facts as set forth by Glickman are confirmed by Leonard who reports on replies received by him in response to 152 letters addressed to teachers of periodontology. In an attempt to remedy the situation he suggests that written and practical examinations in periodontology be included in the state board examinations; that deans of all dental schools be urged to reorganize the whole curriculum so that the objective of dentistry will be made the health and welfare of the patient; and further, that all members of the teaching staff cooperate in presenting their subjects in their relation to periodontal health.

If these recommendations are to be given a place in the dental curriculum it will be necessary to realize that dentistry is more than the sum total of its technical procedures; that the repair and replacement of teeth necessary though that be in daily practice, is not an end in itself but a means to an end and that the maintenance of the mouth and teeth in health is the prime objective of dental education.

As expressed by Glickman, it is not too much to hope "that our dental institutions which have been intrusted with the responsibility for training dentists prove themselves worthy of this trust by having the vision and courage to break away from the hampering tradition of continued subservience to the restorative arts and embark on an educational program which is consistent with the real nature of the dental problem of the population."

A. H. M.

Is Gingival Massage Helpful?

The subject of gingival massage has been discussed frequently by the Council on Dental Therapeutics of the American Dental Association, and was again

considered at one of their recent meetings. The American Dental Association Newsletter in reporting this meeting quoted the Council as follows:

"The merit of the so-called massaging action of the use of toothbrushes has not been scientifically demonstrated. The scientific findings that keratinization of the gingivae may be increased by massage needs to be studied further to determine the relationship to periodontal therapy."

This quotation was printed in the Oral Health News of the Federal Security Agency (Issue 14, March, 1951), and will doubtless be repeated widely as the official opinion of the Council on Dental Therapeutics and the American Dental Association.

It is true, of course, that the value of gingival massage has not scientifically been established to the complete satisfaction of everyone, but the attitude that gingival massage cannot be "approved" will imply to many that dentists should not recommend gingival massage until more scientific evidence is available. There are many patients looking for excuses to keep from using the toothbrush and now they have one: "The Council on Dental Therapeutics says the value of gingival massage is not established."

We believe the tooth brush is a valuable adjunct to the oral care necessary to promote and maintain oral health, and we question the wisdom of releasing any statement which will minimize that value in the minds of patients.

We are tempted to end this editorial at this point, but a sense of fairness compels us to point out that the above quoted statements of the Council are incomplete and do not give a true impression of the opinions of all members of the Council. We have been given permission to quote the complete statement on gingival massage as approved by the Council:

"First, the generally recommended uses of toothbrushes *may* have two aspects: 1) detergent action; 2) massaging action. Second, it is probably safe to assume that the detergent action of the uses of toothbrushes is generally regarded to have value in the promotion of dental hygiene. Third, toothbrushes may be used to meet the definition of massage; i.e., they may be used to rub, stroke, compress or otherwise manipulate the soft tissues of the mouth. However, the merit of the massaging action in the use of toothbrushes has not been adequately demonstrated by controlled studies. The scientific finding that keratinization of the gingivae may be increased by massage needs to be studied further to determine its relationship to periodontal therapy."

We cannot quarrel with this statement; in fact, we believe the Council could have added a comment that gingival massage may be harmful unless properly performed. We are certain that the Council on Dental Therapeutics will not object when we say to the hundreds of dentists who believe in gingival massage that they can continue to recommend it as a beneficial procedure. We can also say to these dentists and men interested in dental research that careful clinical records should be kept and sound research studies should be made to establish the validity of the claim that gingival massage is helpful in promoting and protecting oral health.

An Appraisal of Developments in Periodontology from 1901-50*

By HAROLD J. LEONARD, D.D.S., B.A., *New York, N. Y.*

THE standard text of Oral Pathology and Practice in 1900 was by *W. C. Barrett*[1] published by S. S. White Dental Mfg. Co. in 1898. In discussion of pyorrhea alveolaris and the etiology of loosening of teeth it says, "It has been intimated that the exact nature of the disorder has not been determined, at least no exposition of it has been generally accepted. That its seat is within the alveolar socket is easily demonstrated, and that either the tooth root or its investing membrane is an essential factor in its existence is quite plain, for extraction always affords a radical cure. Beyond this there is no admitted certainty concerning its etiology. . . . We must recognize at least three separate pathological degenerations that are covered by the term, and which, without doubt, are often confounded with each other. The first of these will be entirely local in its character. It will have its origin in an easily comprehensible cause, local irritation. The second will have its etiology in deposits of a hard nodular character upon the roots of the teeth. It will be distinguished by the formation of distinct pockets within the alveolus. The third will give evidence of some distinct cachectic condition or dyscrasia. It will present phenomena which are peculiar to itself, and will be without either of the two previously named factors."

A. W. Harlan in "A Review of Recent Literature of the Loose Tooth or Pyorrhea Problem."[2] (1900) states that the foregoing paragraph in Barrett's text book "may be fairly said to constitute all that a dental student gets in a course of lectures on dental pathology and etiology concerning the loose tooth problem."

Although Harlan's statement was undoubtedly true so far as teaching in the schools was concerned it does not represent the status of the current literature of the time. The subject was much before the profession and many essays on it were read, discussed and published.

As in the case of Medicine, Periodontology has progressed through the technical workers in the field rather than the theorists. Modern medicine is based on the practical work of the barber surgeons and nurses, followed gradually by research experimentalists and has been retarded by the priests and the physician theorists who succeeded them in caring for the sick. So modern Periodontology is based on the work of *John M. Riggs*[3] of Hartford, of *William J. Younger*[4] of San Francisco, *John D. Patterson*[5] of Kansas City, *D. D. Smith*[6] of Philadelphia and others who devised instruments and trained and used their hands to eliminate local irritating factors. They devoted their efforts to scaling all calculus and other deposits from tooth surfaces, to polishing subgingival roughnesses, to planing porous cementum and to teaching patients a meticulous home care technic. *D. D. Smith*,[7] it was who developed the recall routine of the frequent follow up prophylaxis and check on home care. Other technical dentists like *W. G. A. Bonwill*,[8] early in the 1890s introduced the idea that bad articulation is a cause of periodontal inflammation and pocket formation and must be corrected for treatment to succeed, and *W. V-B. Ames*[9] in 1901 demonstrated the value of fixed bridge work for stabilization of loose teeth.

The bulk of periodontal literature of the period, on the other hand, was written by medically trained dentists and dental teachers like *C. N. Pierce*[10] who contended that all pyorrhea, not due simply to salivary deposits, was caused by a uric acid diathesis, or *G. Lenox Curtis*[11] who believed it was caused by syphilis, or *M. L. Rhein*,[12] *Eugene Talbot*,[13] *Henry S. Nash*,[14] *Edward C.*

*Read at the 34th annual meeting of the American Academy of Periodontology October 28, 1950 at Atlantic City, N. J.

Kirk[15] and many others who believed and taught that it was due to various systemic and constitutional conditions. These men wrote and discussed voluminously. The dental literature of the period contained more articles on diseases of the investing tissues and their treatment than any other subject in dentistry excepting pulpal diseases and pulp canal therapy. The diversity of views regarding etiology and the necessity for systemic treatment based on the various supposed systemic causes made treatment by the dentist who was not also a licensed physician seem very discouraging. Ideas, theories and treatment were presented which form the basis of modern periodontology but they were so obscured by the arguing of the theorists that the subject was completely unsatisfactory to the average dentist.

The attitude of average dentists throughout the country may well be illustrated by an incident at the annual convention of the 6th, 7th and 8th District Dental Societies of New York in 1898. After *Dr. G. H. Thompson* of Honeoye Falls had read a paper on Alveolar Pyorrhea and the subject was being discussed,[16] a member, *W. A. Barnes* of Buffalo asked "if any dentist present claimed to cure pyorrhea." None did. *Dr. W. A. Barrows* of Buffalo did not believe the disease could be cured. He said it is a systemic disease finding local expression. The treatment should be both local and systemic. It is easy to remove the calcic deposit but to remove the serumal deposit would endanger the membrane which, if destroyed, will not be recovered. *Dr. F. B. Darby* of Elmira then rose to state that he thought it pretty strong talk for a man to get up in a dental meeting and say pyorrhea could not be cured when we all know what Dr. Riggs did for the disease. He cured his cases in one or two sittings. He knew of one case where the mouth was in a horrible condition, but Dr. Riggs cured it in two prolonged sittings. As to destroying the periostium he did not believe the membrane was in a condition to be of any value where deposits were.

Scientific work in periodontology had already been started at the beginning of the century. *G. V. Black*,[17] in addition to his many contributions in the field of Operative Dentistry, wrote a book on The Periostium and the Peridental Membrane published in *1887*, a histological study of the connective tissue structures and cells of the periodontium. *Vida A. Latham*[18] wrote many articles starting in 1889 on methods of making sections of teeth and periodontal structures for histopathological study. *Eugene Talbot*,[19] in 1899, published his book on Interstitial Gingivitis based on extensive histopathological study of the periodontal structures, including the gingival epithelium, and biochemical studies of calculus. *W. D. Miller*,[20] from 1880 on, included in his researches, studies of the bacteria and fungi of the mouth, focal infection disease, tooth brush abrasion as cause of so-called erosion, mouth antiseptics and other subjects relating to periodontology.

Such researches by individual geniuses continued in the next twenty five years, but research as a recognized part of university discipline in dental schools, as a recognized aim of dentistry and as an important part of dental literature did not get a real start until that great philanthropic scientist, *William J. Gies*,[21] saw the need in dentistry and gave up his life's work to devote himself to the development and elevation of dental research, education and literature. Since the 1920s when the Carnegie Survey of Dental Education was published (1926) and the International Association for Dental Research (1920) with its Journal (1919) was founded, research in dentistry, including periodontology has been stimulated and is increasing each year in volume of output and numbers of people engaged. Whereas in 1900 perhaps a dozen dentists in the United States and Canada were doing scientific dental research, now hundreds are trained for it and are being employed by Universities and other institutions for that purpose. Many are doing it without remuneration

in connection with their private practices. I well remember the times around 1918 when each year a dinner was given for the research workers in dentistry attending the dental teachers convention. *Dr. A. E. Webster,* who was the leading spirit used to have the greatest struggle to get even a dozen members to attend. At the 1949 meeting of the International Association for Dental Research one hundred seventy five members took part and four hundred two persons attended. Of the 131 abstracts presented, 101 were on dental biological researches, of which twelve were purely periodontology while only thirty related to dental materials and drugs. Most of the 101 were devoted to dental caries, histology of tooth structures, biochemistry of teeth and saliva, bacteriology and anatomy. The number devoted to periodontology is as yet relatively small but it is increasing each year as more and more university dental schools develop adequate teaching and research staffs devoted to the subject.

Of the outstanding contributors to dental research in the field of histopathology during the last fifty years, the late *Bernard Gottlieb,* formerly of Vienna, is first, not only for his own findings, but more for the pupils he inspired and started on notable careers. Of these, *Rudolf Kronfeld, Balint Orban* and *Joseph P. Weinmann* have come to the United States and have contributed both in their universities and in the Academy of Periodontology toward building a thorough scientific histopathological foundation in periodontology. Another pioneer is Harold K. Box of Toronto who also has been responsible for starting several men on notable careers. More recently Henry Goldman and Irving Glickman of Boston have come to the fore and are making excellent contributions to our knowledge of periodontal histopathology.

In the field of bacteriology and immunology the research has not been as satisfactory, since much of the work which received prominence for a time proved to be unsound. For many years the various periodontal diseases were regarded as infections due to certain micro-organisms against which research was directed to obtain vaccines, serums or drugs for counteraction. *C. Lenox Curtis*[22] was the leader of a group, especialy in the Army, that believed syphilis was responsible for all or most of the deep pocket periodontal disease. For years various antisyphilitics were discussed in the literature. *Kenneth W. Goadby*[23] of England started in 1904 the use of vaccines. This created quite a vogue that was not checked until 1916 when *Arthur H. Merritt*[24] read a report of research work which fairly discredited vaccines both in theory and practice. A later Inava Endocorps Vaccine was developed by *L. Goldenberg*[25] of the Pasteur Institute in Paris that was supposed to create a local immunity by local injections. This was disproved by a research in 1926 conducted by a committee of the Academy under the leadership of *Isador Hirschfeld.*[26]

In 1914 two pairs of workers, *C. C. Bass* and *F. M. Johns*[27] in New Orleans and *M. T. Barrett* and *Allen J. Smith*[28] of Philadelphia discovered independently an amoeba in the gingival exudate and jumped to the conclusion that they had discovered the causative agent of periodontal destruction. This caused a great furor in dental circles and for some years the literature was flooded with prescriptions for anti-amoebic drugs and reports of benefits. In 1916 *T. B. Hartzell*[29] reported the results of a careful study of treatment with emetin which completely discredited this theory. At that time Hartzell, working with Arther Henrici was at the peak of his fame for his studies on oral bacteria and their relation to focal infection and his findings had great weight.

Infectious conditions about the teeth as a cause of systemic disease have been recognized and noted by writers for centuries. In 1911 the subject was brought forcibly before the profession by a book "Oral Sepsis as a Cause of Disease." published in England by a physician, *William Hunter.*[30] In this book Dr. Hunter particularly castigated American dentists for their crown and bridge work which he contended caused and covered up areas of oral sepsis which

acted as foci of infection for many serious systemic diseases. Such a criticism caused much pain in American dental circles where bridge work was an object of great pride. The National Dental Association (later the American Dental Association) dealt with the matter promptly by appropriating money for a research under the direction of *T. B. Hartzell*[31] to investigate all phases of the oral focal infection problem.

A great deal of very excellent work was done by the group, especially by Arthur Henrici, a bacteriologist and pathologist, a member of the staff of the Medical School at the University of Minnesota. The chief new finding was that teeth with devital pulps, whether or not the root canals were treated, generally had periapical infected granulation tissue near or about the apices. Such masses often become cystic after a time. These masses as well as gingival granulating inflammatory areas, act as pathways by which bacteria enter the blood stream and reach the other body parts where secondary infections were believed to be set up. The chain of evidence seemed to be complete. The salivary streptococci were always present in the local lesions. They could usually be found in the secondary lesion. The patients seemed in most instances to improve promptly when the primary lesions were cleaned up. Animals injected with the bacteria often came down with lesions like those of the patient.

Further substantiation was produced by *Edward C. Rosenow*,[32] bacteriologist of the Mayo Clinic, who contributed evidence of elective localization of the salivary streptococci depending on their adaptation to the oxygen tension of the primary focal infection.

This focal infection theory and research did a great deal of good in that it brought about a reform in operative and prosthetic dentistry procedures toward smooth filling and crown margins and sanitation about the gingivae. Also it challenged the current practice of careless devitalization of teeth and root canal filling and put the burden of proof on the dentist who did such work to show that periapical infection was eliminated.

On the other hand much harm was done in that extremists carried the idea to the point of demanding that their patients have all pulpless teeth extracted whether or not there was evidence of periapical infection.

Clinical experience and subsequent research have not born out the focal infection theory in the way which seemed so definite in 1916. It was found that while salivary streptococci do get into the blood stream from inflammatory lesions about the teeth such bacteremias are evanescent and do little harm, excepting occasionally to heart valves already damaged by anatomical defect or previous disease. Their presence in inflammatory lesions in other body parts is like that of any foreign particles in the blood stream which tend to collect in any area of inflammation. Patients who improve so markedly after removal of primary foci, generally have recurrences when away from hospital care. Many rheumatic cases improve in a manner similar to that of removal of foci of infection when any operation is performed, even though it has nothing to do with infection. The removal of foci of infection, especially around teeth, as a cure for many systemic diseases, regarded for several years both in medicine and dentistry as the long sought panacea for rheumatism and a host of other diseases of unknown etiology has proved to be largely illusory. The present thought seems to be that periodontal inflammation whether gingival or periapical, especially if extensive, may contribute to ill health and disease in other body parts, but such focal infection is only one of many factors such as dietary deficiencies, endocrine imbalances, constitutional predispositions, allergies and psychosomatic influences. Dental foci of infection should be eliminated but no promises should be made that such elimination will permanently cure the rheumatic or other systemic condition the patient may have.

The trend in periodontology is away from the conception of gingivitis and perio-

dontitis as primarily infectious diseases. Present thought seems to incline to regard periodontal disease as inflammation due to local irritants and influenced by physico-chemical conditions in the tissues and not as infection in the usual sense of that term. The bacteri and fungi which grow on the teeth are important for they cause irritation and inflammation of the soft tissues against which they lie and the inflammations lead to the degenerative changes and destructions resulting in pocket formation. Occasionally when the periodontal tissues are injured deeply by trauma from faulty occlusion, localized infections may occur in such injured tissues leading to periodontal (pericemental) abscess. For the most part however it appears from the researches, especially of *Theodore Rosebury*,[33] that the overgrowths of certain of the mouth bacteria and the reactions of the tissues to the presence of these micro-organisms are due to physico-chemical conditions in the gingival tissues rather than to variations in kind or virulence of mouth micro-organisms. Even the so called Vincent's gingivitis with its rapid onset and fulminating character is due to reaction from overgrowth on the tooth surfaces of habitual mouth organisms, such overgrowth and such reaction being in turn due to temporary alterations in the chemistry of the tissues induced by one or more local or systemic causes not yet fully defined.

This concept of the role of bacteria in diseases of the gingiva and deeper periodontal structures is not alone the result of the researches of bacteriologists, but has been gradually developed by the careful study of clinicians who have observed and compared results when hygienic measures have been met or not met. These measures consist in keeping bacterial growth to a minimum by sanitary mouth procedures, keeping gingival tone high by massage, masticatory function and freedom from mechanical injury, and by keeping the general health good by general personal hygiene and optimal nutrition.

This concept, in my opinion, has been the greatest single factor in bringing the subject of periodontology into perspective. It has stopped the wild goose chase after a specific micro-organism causing periodontal disease or after a specific vaccine or drug to counteract it. It has placed the emphasis where it belongs on hygiene; cleaniness and freedom from irritation of the periodontal structures, maximum local vigor of these tissues, and maximum general health and optimal nutrition of the individual as a whole. The concept is not invalidated by the fact that we can not yet identify all the factors, especially the systemic and constitutional ones that create chemical conditions in the tissues which invite bacterial overgrowths on the teeth or untoward reactions to such growths. There is still much to be learned about the biochemistry of the gingival tissues and the exudates therefrom as influenced by heredity, systemic disease, nutritional deficiencies, poisons, endocrine imbalances, allergies and psycho-somatic factors.

A modification of the idea that bacterial growth on tooth surface is the active cause of gingival and periodontal breakdown has been introduced by *Gottlieb*[34] and is especially emphasized by the histopathologists. It is the idea that one form of periodontal disease (periodontosis) leading to great loss of alveolar bone and to deep pockets occurs without appreciable initial inflammatory changes. It is primarily a degenerative change in the periodontal membrane accompanied by failure of new cementum formation and by loss of alveolar bone. This degenerative change permits deep penetration of gingival epithelium along the root which in turn may become separated from the root to form a greatly deepened sulcus into which bacteria grow with accompanying inflammation later on. The degenerative change is assumed to be due to some constitutional or systemic factor because from post-mortem material it has been impossible to discover possible local factors. So far no correlation has been noted with connective tissue degenerative changes in other body parts. Nor has the condition been definitely related to an endocrine or other systemic

dysfunction. It is one of the unsolved problems at the present time. Fortunately such cases are infrequent in practice so that they do not create a reason for pessimism in the treatment of periodontal diseases as a whole.

It would appear at present that research devoted to biochemical pathologic changes in periodontal tissues correlated with similar changes in other body parts should be even more productive than further histopathologic or bacteriologic work. Some work has been done on the general biochemistry of bone physiology and pathology by *Leriche and Policard*[35] (1928) and on the chemistry of inflammation by *Valy Mencken*[36] (1920). *Harold Box*[37] and his coworkers in Toronto have applied the work of Mencken to the periodontal tissues with results which are interesting and may prove very significant. One finding is that injured cells may give off a chemical called necrosin which has the property of causing death of tissue along the lymph channels of the periodontium where it is draining. This finding raises the question whether the deep degenerative changes found in so-called periodontosis may not after all turn out to be due to some such local mechanism.

Another biochemical study has been that of the late *Daniel E. Ziskin*[38] and his associates on gingival changes due to endocrine imbalances. He found that estrogenic hormones promote a well developed oral epithelium while deficiency of these hormones or excess of their antagonist folluitin leads to gingival epithelial changes characterized by loss of resistance to infection and loss of outer keratin layer. Such conditions he found in certain cases of pregnancy leading to pregnancy gingivitis and in some elderly women and men leading to a condition called desquamative gingivitis.

A great deal of research has been conducted by dentists and others on nutritional deficiencies in relation to oral disease. Most of the dental research of this nature is especially related to the cause and prevention of dental caries, but some is related to the periodontium. *Percy Howe* (1920-1942)[39] followed by *Eddy and Dalldorf*[40] (The Avitaminoses, 1937) and *Paul Boyle*[41] (1933-1950) have studied the periodontal effects of vitamin C deficiency (scurvy) both in their aggravated and subclinical forms. *Hermann Becks*[42] and his coworkers have also conducted much excellent research on the periodontal effects of deficiencies of vitamin B complex components.

Periodontists individually and collectively through the Academy have been intensely interested in nutrition, probably more than any other group in Medicine or Dentistry excepting the pediatrists. They have seen that in localities where dietary conditions are optimal that dental disease is negligible and other diseases, including cancer reduced to a minimum. They are convinced by their clinical experience that susceptibility to periodontal disease is very largely a matter of nutrition of the periodontal tissues which it seems logical to suppose is directly related to the diet. Research[43] on experimental animals has shown that periodontal disease can be produced or prevented at will by changes in proportion of nutrients. The whole philosophy of periodontology causes its emphasis to be placed on hygiene, creation and maintenance of health rather than on the therapeutics of disease. Periodontists have gone far in the first two phases of their effort, (1) the creation of sanitary conditions and freedom from irritation about the teeth, and (2) the creation of vigor and tone in the periodontal tissues by stimulating circulation with local, home care massage and by causing each tooth to function, brought about by vigorous mastication on teeth maintained in or restored to good occlusal equilibrium. They therefore struggle toward the third step, the establishment and maintenance of a general health and optimal nutrition created mostly through adequate diet that will do for their patients what such nutrition has done for certain isolated groups studied by *Robert McCarrison*[44] (1921) *Weston A. Price*[45] (1939) and others.

The efforts to bring about general health as a means of creating and maintaining dental health have not been very satisfying up to this time. Physicians are primarily interested in the therapeutics of disease even more than the diagnosis of disease and very much more than in maintaining a good state of health. This is due partly to faulty emphasis in their education but more to the exigencies of their practice. The difference in interest and emphasis makes cooperation of the physician with the periodontist unsatisfactory. The periodontist recognizes from the reactions of the periodontal tissues that a nutritional fault in those tissues is preventing successful treatment. Believing that such fault must be of systemic and possibly dietary origin, he refers the patient to the physician to discover what it may be and to get help in correcting it. The physician, not finding frank systemic disease by his routine tests, and not being interested or educated in the means for maintaining optimal health, is unable to help.

Nutrition research has not been as rewarding in the periodontal field as could be desired. Animal research has been directed for the most part to feeding diets deficient in one known nutrient but adequate in all others, to determine the role of the nutrient deficiencies in creating disease. Such research has not produced results of much clinical value for periodontists. Severe deficiencies of some nutrients may lead to oral disease which may be recognized, diagnosed and treated by diet correction by the periodontist. Such cases however are rare in the average periodontist's practice.

Even when dietary experiments on animals have been directed to creating optimal health and longevity the results have been difficult to apply to human patients. In the first place, human nutritional requirements are not exactly the same as those of any experimental animal. In the second place it is difficult to get people to change their dietary habits and to use the foods that have been proved effective for animals. Also the conditions of commercial farming, processing, storing, distributing and preparing food stuffs alter foods to such an extent that what might be an ideal food under natural conditions might be very faulty as eaten at the dining table. Wheat, for instance, raised on good soil is an excellent and highly protective food for rats in its raw or fresh finely ground state. As prepared for humans in the form of bakers white bread it is far from a protective food and there is even debate as to whether it is not poisonous. Add to this the misleading advertising of the food processors and the pseudoscientific books, articles and lectures of food faddists and the whole subject becomes highly confusing.

What is needed is research starting with the diets which have been found to be ideally suited to human nutrition and have furnished optimal health, vigor, longevity and freedom from disease. Such diets have also been shown to be ideally suited to animal nutrition. From these diets substitutions should be made with foods as eaten by modern human beings until it is discovered what combinations of foods available in the markets and prepared by current methods are optimal. If changes must be made in methods of farming, processing, storing, distributing and preparing foods in order to maintain valuable properties these should be determined and definitely stated so that concerted effort can be made to have them changed. When the facts are definitely determined as to how optimum nutrition can be attained with the least amount of disruption of dietary habits and least waste of money, then periodontists can add education in diet and general personal hygiene to their other efforts. Until these facts are shown this phase of periodontia practice must remain in an unsatisfactory state.

The technics of periodontia have been modified to some extent in the half century. *Riggs*[46] taught a thorough scaling technic with a vigorous curettage of the soft tissues at the base of the pockets or sulci. *Talbot*[47] also taught careful scaling of unattached tooth surfaces but advised against

any injury of the soft tissues of the gingival wall or epithelial attachment at the base of the pocket. These differing methods have their protagonists to the present day. The advocates of curettage of the soft tissues at the base of pockets and inflamed sulci have not conclusively demonstrated a reattachment different in degree or kind from the closing of the pockets by close adaptation usually following good treatment without such soft tissue curettage. Nor have they shown that health supervenes more rapidly because of the acute inflammation induced by the surgical interference than occurs when the tissues are stimulated by gingival massage alone. The evidence to date seems to favor the use of the gentler method.

About 1918 surgical resection was introduced into the United States. The method had long been advocated in France (*Fauchard* 1728 and *Bourdet* 1775)[48] but seems not to have been used in the United States prior to the First World War. The method was adopted and vigorously taught by *Arthur Ward*[49] and *Arthur Black*[50] and subsequently by *Olin Kirkland*.[51] Ward invented an elastic antiseptic cement or pack which he applied to cover the cut surfaces to prevent after pain and infection and the growth of exuberant granulations until healing had occurred.

The operation, as generally performed, removes the gingival tissue as far as the pocket extends, after which the wound surface is vigorously curetted to resistant bone or dense connective tissue. This removes all epithelial attachment and all bits of soft inflammatory tissue. The root surfaces are also curetted at this stage under direct vision. If the detachment extends below the border of remaining process the bone is trimmed so that a trough will not be left. The raw surfaces are then covered with a pack, which covering is maintained for at least two weeks. By this time epithelium has grown over the wound surface to the root to which it attaches. There is some evidence that this attachment grows up on the root to a level as crownward as previously or even more so. After a few months a new gingiva grows up about the root which is free of the old inflammation and congestion providing sanitation has prevented any new irritation. Usually the sulcus depth remains small. The method causes a temporary loosening of the affected teeth because of injury to the transverse periodontal fibers and a much greater permanent gingival recession than usually results from the so called conservative treatment. It has the advantage that it gets rid of the deepened sulcus or pocket which, even with the best of home care almost inevitably tends to collect bacterial and fungous slime and cause recurring inflammation, exudation and calculus formation. Such redepositions require the intervention of expert periodontal treatment at twice yearly intervals or oftener to maintain a healthy gingiva.

The present practice among those who are highly skilled in both the conservative and surgical resection methods is to use both methods, the choice being indicated by a number of factors having to do with the patient's desires and circumstances as well as the location and depth of pockets in the mouth. By recognizing the advantages, disadvantages and limitations of both methods, an intelligent choice can be made which will be most likely to meet the health needs of the case and the desires of the patient.

The tendency of the last fifty years has been away from the use of drugs in the treatment of periodontal diseases. In the beginning, because the causative agent was believed to be one or more micro-organisms, the search was for a drug that would kill out or decrease growth of such micro-organisms or, on the other hand, alter the tissue tone in some way to raise its resistance. This was a dental counterpart of the search in medicine for drugs that would cure or aid in the cure of disease whether or not the cause of the disease were known or the pharmacologic action of the drug. When the patient is sick or in pain there is a demand on the doctor to do something. If he does not know what will really help he can at least give a drug that will satisfy

the patient's demand and help psychologically if nothing more.

As periodontists have come to realize that gingivitis and periodontitis are caused primarily by oral insanitation they have come to depend on hygienic measures and to eliminate drugs almost entirely. To a modern student of the subject a dentist who tries one drug after another in an attempt to cure gingivitis, Vincent's infection, or periodontitis is just fifty years behind the times. There are, however, some drugs which have special uses in aid of the periodontal operative procedures. Local anesthetics are much used, especially for surgical procedures. Frothing oxidyzing agents such as hydrogen peroxide or sodium perborate are useful to boil out sulcus debris painlessly in acute Vincent's cases and reduce all symptoms to the point where instrumentation can be done in a day or two without undue discomfort.

Reliance on penicillin as a cure for gingivitis, Vincent's gingivitis or periodontitis, it seems to me, is a return to the outmoded approach. Penicillin injections to a point of high blood level will cause bacterial growth in gingival sulci to decrease and thus cause an improvement in local symptoms. When the penicillin blood level goes down the bacterial growth recurs together with the symptoms. Nothing permanent has been accomplished. Penicillin troches also cause temporary subsidence in bacterial growth on the teeth and in sulci which the drug can reach. They are thus much used for acute Vincent's gingivitis. The temporary effect is no better than that of the much less expensive hydrogen peroxide debridement. Many people quickly develop an oral allergy to penicillin troches and get an acute generalized stomatitis that may be very painful and last for some days. Very few ever develop such an allergy to a short course of hydrogen peroxide mouth irrigation. It seems to me that the use of penicillin in gingivitis of any kind is not in accord with modern periodontology thinking. On the other hand the use of penicillin injection for the abortion of acute abscesses, even periodontal (pericemental) abscesses, may be a method of choice in handling such cases. The foregoing applies as well to the use of other antibiotics.

Drugs may have some use in the reduction of body odors. Chloramine T has been put up as Aromatic Chlorazene powder which is very useful when made up in 2% solution in destroying odors emanating from the mouth. It is particularly good as a mouth wash in cases such as an acute leukemia where gingival bleeding causes a horrible mouth odor. Chlorophyllin, now dispensed in tablet form for ingestion, is useful in destroying the body odors of elderly or sick people, which odors often appear in the saliva and cause a very fetid breath. These odors are apparently caused by cholesterol and other sterols which the chlorophyl helps to counteract and eliminate.

When the Academy of Periodontology was organized in 1914 it was named "The American Academy of Oral Prophylaxis and Periodontology" with the idea that the prevention of dental caries was a purpose and responsibility equal in importance to the Academy with the study and treatment of the investing tissues. As time has gone on this attitude has changed and the study of dental caries and its prevention has been eliminated as the direct responsibility of the Academy and informally delegated to the research workers in dentistry and to a lesser extent to public health dentists. At the time the Academy was formed such groups had not yet come into organized existence.

It is well that this study has been assumed by others for its ramifications are great and lead far afield while the study of the periodontal and soft tissues of the mouth, including bone, and their relations to systemic disease and hygiene including nutrition has become sufficiently large to engage the entire scientific attention of the Academy. It is very possible however that much of the etiology and prevention of dental caries will be found to be related to the chemistry of gingival exudates which

are definitely in the field of periodontology. Conversely it may be found that deposits on the teeth which are so important a cause of gingival irritation may be explained by studies of the chemistry of saliva which are chiefly on the agenda of dental research.

The fact that periodontal practice has developed along the line of hygiene with its goal the sanitation and optimal health of the teeth, periodontal structures and the patient as a whole, has made it somewhat outstanding in the medical and dental fields and in advance of general medical thought. This channeling is due, I am sure, almost entirely to the American Academy of Periodontology and to the direction given it by its founders, *Gillette Hayden* and *Grace Rogers Spalding*. They, in turn, got their direction of thought from *D. D. Smith* of Philadelphia, known as the "Father of Oral Prophylaxis."

The Academy, also due to the direction given it by its founders, has gained a very much respected position among dental organizations. Its fine scientific efforts, its excellent educational work in preparing popular bulletins and newspaper articles, its freedom from political taint, its opposition to commercialism in professional relations, its efforts to advance higher standards of post graduate education for periodontists and all dental specialists through the American Board of Periodontology and the Advisory Board for Dental Specialties, its very high type of professional journalism, its efforts to promote oral hygiene, oral diagnosis and periodontology education for undergraduates in dental schools, and for graduate dentists through the Sections in the American Dental Association, and above all for the fine gentle fellowship at its meetings, have made it recognized as superior among dental societies. Periodontology is no longer openly sneered at or thought of slightingly. Periodontists are coming to be respected as experts who can do what they say.

There are some things the Academy could do to increase the respect in which periodontology is held by dentists in general. One of these is to simplify or eliminate classification of periodontal diseases, at least until much more is known as to pathology and etiology than at present. The attempt by which each periodontology teacher tries to build an elaborate original classification when the bases for such classifications are not established, simply confuses students of the subject and makes Periodontology appear slightly ridiculous.

Another improvement would be to slow down the attempt to create a new nomenclature until the facts are surely established on which terms can be based. Some of the terms coined by the Academy in the past are so foreign to dental usage as to sound bizarre. The zeal of nomenclature committees each year to perform a superlative job needs to be curbed and only such terms adopted as are necessary, are based on proved fact and are the simplest possible for the purpose.

The status of undergraduate education in periodontology is still very unsatisfactory although far better than that at the turn of the century. Of the forty seven dental schools in the United States and Canada, twenty three have periodontology staffs having at least one well trained periodontist and more than one teacher. The remainder do not have sufficient staff including a qualified periodontist to do any kind of a job at training undergraduates adequately in this branch of dentistry. Many schools assign the teaching of periodontology to a teacher of oral pathology who has no particular training or interest in practical periodontia. Such an assignment seems to cover the matter from the standpoint of the bulletin, but is a dishonest sham so far as real education and training in this important branch of dentistry is concerned. This situation is not always due to a lack of desire on the part of school authorities. Some of the schools are located in cities where no qualified periodontist is practicing and available as a part time teacher. Until recently periodontists having an adequate postgraduate education and training have

not been available as teachers in this field. The very recent development of graduate and post graduate periodontology facilities stimulated by the Board of Periodontolgy and the Council on Dental Education is rapidly filling this vacuum.

Of the twenty three schools with at least a minimum of staff I consider that not more than nine have staffs of sufficient number and competence to man a department or division of university grade involving undergraduate teaching on a scientific level, expert technical training, sound research and competent graduate work.

The difficulty is not all with the staff of periodontology. This subject, being a comparative newcomer in the dental curriculum, must force its way into a course of study already over crowded because of the demands of technical teachers for time for practice in the technical procedures of operative and prosthetic dentistry. The result is a time allotment for periodontology, oral diagnosis and oral hygiene which is quite insufficient to develop the scientific education, habits of thought and technical practice necessary to make students feel confident in these subjects when they graduate. To correct this situation I think a reorientation in dental education will be required. The curriculum of courses of study will need to be revised to make health the end and aim of dental education and dental procedures. Fundamental science courses and all dental courses will need to be revised to eliminate wastes which do not contribute to this aim. The prevention of dental and other diseases and the establishment and maintenance of health, beauty, comfort and function of the teeth, jaws, mouth and body as a whole so far as this can be accomplished through dentistry, should be the objective of dental education. Perfection in the technical procedures in dentistry is a means to this end. Technical procedures should never be allowed to appear as an end in themselves. Such a revision in aim and method would place oral diagnosis, oral hygiene and periodontology in the forefront of dental education with every other scientific and technical subject designed to support them. In this way these subjects would get their proper emphasis and students would get adequate preparation in them without necessarily requiring a much greater allotment of hours devoted primarily to them than at present.

As I review the progress of the last fifty years in periodontology I think there is reason for a great deal of satisfaction. Scientific understanding of periodontal diseases is not yet complete but it is sufficient so that, with very few exceptions, qualified periodontists can restore health to the teeth and periodontal structures by their procedures and maintain it for the lifetime of the patient. Interest in periodontology in keen and increasing so that sessions of dental meetings devoted to it are as well attended as any. The subject has developed in the dental schools so that practically every dental school has, at least on paper, teachers devoting themselves to it. More and more schools are building departments capable of work of university grade in this field. From these are coming highly trained young men and women, well prepared for staff positions, for research work and for expert practice. In spite of increasingly arduous requirements a growing number, now at least a dozen, of qualified candidates appear each year for certification. Each year more conferences lasting several days and devoted to post graduate periodontology and allied subject matter are being put on by dental colleges or societies at Ann Arbor, Palm Springs, the Berkshires the Poconos and elsewhere. Each year it becomes easier for periodontists to work with general dentists, prosthetists and orthodontists because the relationships between faulty occlusion and periodontal disease and conversely between periodontally weakened teeth as related to bridge and denture stability become better understood by both periodontists and others, and because periodontists are becoming recognized as having expert services and opinions to contribute.

The Academy can congratulate itself for these developments for they have not come

by chance or in the general course of things but only by the hard devoted work of this Academy, its Committees, its Board and its individual members whom it has inspired. Such developments have not come about in England or France or other countries where they do not have an Academy based on a concept of hygiene and inspired by such founders and devoted workers as Grace Rogers Spalding and Gillette Hayden.

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American Academy of Periodontology

Annual Spring Seminar

W. K. Kellogg Foundation Institute

at Ann Arbor, Michigan

April 30, May 1, May 2, 1951

An excellent program for the annual spring seminar of the American Academy of Periodontology has been arranged and is enclosed. This will be held in Ann Arbor, Michigan, on April 30, May 1, 2, 1951. All members of the Academy are eligible to attend, and the arrangement committee feels that the program should justify a good attendance. The fee for the course is $25.00 and payable with the application or at the time of registration. Those planning to attend this meeting should write to Dr. William R. Mann, W. K. Kellogg Foundation Institute: Graduate and Postgraduate Dentistry, Ann Arbor, Michigan. The applicant must also write to the Michigan Union, Ann Arbor, Michigan, and make a room reservation giving the time of arrival and departure. Make mention of the fact that you are planning to attend the seminar of the American Academy of Periodontology. We are not permitted to make reservations for you.

Guests may attend the meeting if sponsored by a member of the Academy. Each member is entitled to one guest.

Monday, April 30

9:00 Registration

10:00 Dr. Harry Sicher—"Eruption and Physiologic Movement of Teeth"

11:00 Dr. Gustav Rapp—"Bio-Chemistry of Enzymes"

LUNCHEON

1:30 - 4:30 Dr. John W. Robuck—"New Concepts of White Blood Cell Function in Inflammation as Observed in Human Windows and Some Observations on Alterations Produced by ACTH"

Tuesday, May 1

9:00 Dr. Harry Sicher—"Functional Action of Temporomandibular Articulation"

10:00 Dr. Gustav Rapp—"Bio-Chemistry of Calculus"

11:00 Dr. Rene Rochon—"What to Look for and Where to Look for It"

LUNCHEON

1:30 - 4:30 Dr. Raymond W. Monto—"Hematologic Diseases and Their Oral Manifestations"

6:00 COCKTAILS AND DINNER

Wednesday, May 2

9:00 Dr. Harry Sicher—"The Problem of Attrition and Traumatic Occlusion"

10:00 Dr. Gustav Rapp—"Theories of Preventing Calculus Deposition"

11:00 Dr. Rene Rochon—"The Role of Function in the Interpretation of Radiographs"

12:30 FINIS

APPLICATION BLANK

Name .....

Address .....

Are you a member of the Academy? ..... Yes .... No ....

Reservation has been requested at the Michigan Union or otherwise planned for? ..... Yes .... No ....

Mail to Dr. William R. Mann,
W. K. Kellogg Foundation
Ann Arbor, Michigan

Announcements

Periodontal Disease Workshop

Ann Arbor, Michigan, September 10-15, 1951

The faculties of the University of Michigan School of Public Health and School of Dentistry jointly have planned a third dental inservice training course. The theme of the first one was dental health economics and that of the second one was dental caries control technics. This third dental workshop will attempt an evaluation of the present accurate scientific information concerning normal periodontal tissues, along with the etiology, pathology, symptomatology, prevention and treatment of the various types of gingivitis and periodontal disease. The period selected for this workshop is September 10 through 15, 1951.

This week-long conference will utilize the workshop technic of consultant and group participation—a technic thoroughly tested previously as a means to determine fact. During the first day of the program, and the four succeeding morning periods, lectures will be presented by well-known researchers and clinicians who will attempt critical appraisals of current information about oral histology, physiology and pathology and the etiology, diagnosis, prognosis and treatment of periodontal disease. The references used by each essayist will be duplicated for distribution at the time of his lecture. Four afternoons and the concluding day of the conference will be devoted to small-group determinations of the validity of information in specific areas of the congeries of conditions that are reflected in the health or the disease of the dental investing tissues.

The evaluating groups will be arranged through advance correspondence by self selection of the participants. Their discussion and library sessions will be guided by highly qualified leaders; an enlarged library staff will be available to secure references promptly; and the staff members of the Department of Public Health Statistics will be available to check the validity of data should the occasion arise.

The five areas of information to be appraised by these evaluating groups follow:

I. Behavior of Gingival and Supporting Tissues

II. Local Factors in Periodontal Disease

III. Systemic Factors in Periodontal Disease (In Gingivitis, Vincent's Disease, Advanced Periodontal Conditions)

IV. Treatment of Periodontal Disease (Conservative, Surgical, Occlusal Equilibration)

V. Prevention of Periodontal Disease

Five sets of controversial and fact-finding questions have been assembled to assist the evaluating groups during their study periods.

Further information and application blanks can be obtained by writing Kenneth A. Easlick, University of Michigan, School of Dentistry, Ann Arbor, Michigan. A registration fee of $25.00 will be collected.

Academy of Periodontology to Sponsor Essay Contest

Plans were approved at a recent meeting of the Council of the American Academy of Periodontology for two essay contests to be held for undergraduate dental students each year. Details of these contests have already been sent to the deans of the various dental schools in the United States and Canada. Briefly the rules are:

A. Local Contest. The best essay on some phase of Periodontia that is written each year by its undergraduate students is to be selected from each dental school in any manner that its faculty desires. The name of the winner is to be submitted to the Editor of the Journal of Periodontology before May 1 of each year, and the winner will receive a year's subscription to the JOURNAL OF PERIODONTOLOGY.

B. National Contest. The winner of each local contest is entitled to enter a national contest if he so desires. Papers are to be submitted to the Editor of the JOURNAL OF PERIODONTOLOGY before May 1 of each year. The Editor will then submit the papers to the Undergraduate and Postgraduate Committee of the Academy for judging. Annual prizes will be $50, $25, and $10.

It is hoped that this project will promote interest in periodontia in the dental schools.

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Columbia University

Three postgraduate courses in Periodontology are offered at Columbia University, School of Dental and Oral Surgery of the Faculty of Medicine.

A full time, two academic year program leading to a Certificate of Training. Correlation of the fundamental sciences to clinical practice is emphasized.

A one-half day each week course from September 26, 1951 to May 19, 1952 and a two week, full time course from June 2-13, 1952.

For further information write to Dr. Barnet M. Levy, Director, Postgraduate Division, School of Dental and Oral Surgery, Columbia University, 630 West 168th Street, New York 32, New York.

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The Second Annual Berkshire Conference in Periodontology and Oral Surgery

Tufts College Dental School will conduct the Second Annual Berkshire Conference in Periodontology and Oral Pathology at Eastover, a resort hotel in Lenox, Massachusetts, June 12 to 16, 1951. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities, with afternoons open for recreation. A special activities program is scheduled for the ladies. For further information regarding this conference write:

The Berkshire Conference
Tufts College Dental School
136 Harrison Avenue
Boston 11, Massachusetts

Special Announcement

New York University College of Dentistry

This school offers a one academic year full time course covering all phases of periodontal diagnosis and treatment, including conservative and radical techniques and equilibration of occlusion. Each registrant must acquire sufficient clinical experience to prove his skill in the handling of all phases of periodontal disease. He is also required to prepare an original thesis on a phase of the subject, this is to be presented before the class for discussion. Courses in periodontia are under the supervision of Professor Samuel C. Miller.

One academic year, daily, Monday through Friday, 9 A.M. to 4 P.M. Limited to 4. Tuition $1,000.00. Books $29.50. Instruments approximately $100.00. Rental fee $1.00.

For information and application write to Secretary of Postgraduate Division—New York University College of Dentistry—209 East 23rd St., New York 10, New York.

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The Ohio State University College of Dentistry

Postgraduate Division

The Ohio State University College of Dentistry Postgraduate Division announces Postgraduate Courses in Dentistry for the spring quarter, 1951, including one in Periodontia April 30 - May 4, by Dr. J. R. Wilson.

This course is designed to review the basic principles of periodontal disease and to develop a rational approach to treatment. The course is presented as a series of lectures, discussions and clinical demonstrations. The disturbances on the periodontium, classification of periodontal disease, diagnosis, prophylactic therapy, conservative treatment, surgical therapy, home care, maintenance of periodontal health and related stomatitis will be included. The emphasis will be on proper diagnosis, adequate treatment and essential maintenance.

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University of Minnesota

Fellowships and Teaching Assistantships are available for a limited number of graduate students in the University of Minnesota School of Dentistry. Stipends for these appointments range from $1200 to $3600 per year and include tuition exemption. Graduate study programs may be arranged with a major in Orthodontics, Oral Pathology, Oral Surgery, Oral Medicine, including Periodontia or Restorative Dentistry (Operative, Prosthetics, Pedodontia, or Endodontia.) The courses in these areas vary in length from two to three years. An alternate program, in the basic sciences with special references to dentistry, leads to the Ph.D. degree.

Persons interested in these appointments should write for an information bulletin and application form to the Dean of the Graduate School, University of Minnesota, Minneapolis 14, Minnesota.

Letter to the Editor

I was much interested in and, in some respects, in disagreement with statements in some of the articles published in the January 1951 issue of the JOURNAL OF PERIODONTOLOGY. As the points with which I disagree are fundamental to the further development of a system of periodontal pathology, I think it important that I present my views to the various authors and to periodontists in general for what they may be worth.

The most important matter is: What is and what is not periodontosis? The term "periodontosis" is used freely in these papers (by Schour, Marshall-Day, Kerr, Bernier) with the implication that (1) it is a clear-cut pathologic entity, and (2) a non-local, degenerative etiology has been established for it.

Since none of these authors has felt it necessary at this time to describe what he conceives periodontosis to be, I will insert here a description which embodies commonly-held ideas: Periodontosis is a lesion involving characteristically one or more of the upper incisors, the manifestations of which are labial wandering with resulting diastema and either absence of periodontal pocket or pocket formation without pus (sic). It is supposed, as I indicated before, to be degenerative and thus of systemic origin and is believed not to have a local etiology such as traumatic occlusion and/or calculus formation.

If this is a distinct disease and if it is solely of systemic origin, I ask the question: Why does it affect only the upper incisors? I have never seen it identified as a disease which also affects other teeth in the way described.

Now, I agree that there is such a disease as periodontosis, or at least that it is a convenience to have such a name to apply to a fairly clearly marked pathologic entity. Periodontosis (i.e., as I see it) is a degenerative disease and undoubtedly of systemic origin. But my conception of periodontosis is that it is a disease that attacks quite early in life, characteristically at or near puberty (I have seen quite a few cases at the Guggenheim Clinic) and that it is manifested first and most severely in the first permanent molar region, jumping from there to the incisors and ultimately involving all other teeth. Karshan, in his paper in the same issue of the JOURNAL, gives a brief description of the pattern of bone loss. (I should say that while he mentions diastemata, and I agree that these sometimes occur, they are in my observation late manifestations.)

As to the cases in which diastemata are found in the upper incisor region only and they make their appearance only in the middle-aged as commonly found, my position is that these cases are not cases of periodontosis but of periodontitis.

I have one more "bone to pick." That is the occasional improper use of the term "periodontitis," as, for example, "periodontitis proper" (Schour). Here it is proposed to use the term to designate as inflammatory condition originating in and, for a time, presumably limited to the periodontal membrane.

When the Academy was organized I undertook to develop at least the beginning of a nomenclature. I proposed the term "periodontium" as an inclusive term to designate the tissue complex consisting of gingiva, pericementum, and alveolar bone. Thus "periodontitis" is an inflammatory condition attacking all three of the supporting structures of the teeth. I do not feel that it is proper to limit it to just one of these tissues. The term "periodontitis proper" thus either is supererogatory or incorrect.

If it is felt necessary to coin a term to designate the condition to which Schour applies this designation, I suggest "lateral pericementitis." It is, of course, well known that there are two main sources of pericementitis, (1) periapical infection, from an infected pulp canal, and (2) periodontal, arising in gingivitis, etc. If it is proper to call a periodontal abscess a lateral abscess, it would seem to be satisfactory to call the condition Schour describes as lateral pericementitis. Periapical pericementitis would then be the designation for the inflammation arising at the root apex.

John Oppie McCall, D.D.S.